



RESEARCH ARTICLE

Joint modeling the frequency and duration of accelerometer-measured physical activity from a lifestyle intervention trial

Juned Siddique¹  | Michael J. Daniels² | Gül Inan³  | Samuel Battalio¹ | Bonnie Spring¹ | Donald Hedeker⁴

¹Department of Preventive Medicine, Northwestern University, Evanston, Illinois, USA

²Department of Statistics, University of Florida, Gainesville, Florida, USA

³Department of Mathematics, Istanbul Technical University, Istanbul, Turkey

⁴Department of Public Health Sciences, University of Chicago, Chicago, Illinois, USA

Correspondence

Juned Siddique, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, 680 N. Lake Shore Drive, Suite 1400. Evanston, IL 60611, USA.

Email: siddique@northwestern.edu

Funding information

National Heart, Lung, and Blood Institute, Grant/Award Numbers: R01 HL075451, R01 HL158963; National Institute of Diabetes and Digestive and Kidney Diseases, Grant/Award Number: R01 DK125414

Physical activity (PA) guidelines recommend that PA be accumulated in bouts of 10 minutes or more in duration. Recently, researchers have sought to better understand how participants in PA interventions increase their activity. Participants can increase their daily PA by increasing the number of PA bouts per day while keeping the duration of the bouts constant; they can keep the number of bouts constant but increase the duration of each bout; or participants can increase both the number of bouts and their duration. We propose a novel joint modeling framework for modeling PA bouts and their duration over time. Our joint model is comprised of two sub-models: a mixed-effects Poisson hurdle sub-model for the number of bouts per day and a mixed-effects location scale gamma regression sub-model to characterize the duration of the bouts and their variance. The model allows us to estimate how daily PA bouts and their duration vary together over the course of an intervention and by treatment condition and is specifically designed to capture the unique distributional features of bout PA as measured by accelerometer: frequent measurements, zero-inflated bouts, and skewed bout durations. We apply our methods to the Make Better Choices study, a longitudinal lifestyle intervention trial to increase PA. We perform a simulation study to evaluate how well our model is able to estimate relationships between outcomes.

KEYWORDS

gamma mixed-effects location scale model, joint modeling, poisson hurdle, zero inflation

1 | INTRODUCTION

1.1 | Physical activity interventions

The 2008 physical activity (PA) guidelines for adults from the U.S. Department of Health and Human Services recommend at least 150 min a week of moderate-intensity PA, 75 min a week of vigorous-intensity PA, or an equivalent combination of moderate-to-vigorous intensity physical activity (MVPA). According to the 2008 report, to meet these guidelines, MVPA must be accumulated in bouts (episodes) of at least 10 minutes in duration.¹ Unfortunately, less than 50% of

TABLE 1 Descriptive statistics on zero-bout days, non-zero bouts, and bout duration by study phase and treatment condition in the MBC study.

Phase (length)	Treatment Condition	Percent days Zero bouts	Mean (SD) Non-zero bouts	Mean (SD) Bout duration
Baseline	Increase PA	66.7	1.83 (1.08)	12.9 (8.8)
(2 weeks)	Decrease SED	72.3	1.79 (1.04)	14.0 (9.3)
Rx1	Increase PA	51.1	1.88 (1.07)	16.6 (10.5)
(1 week)	Decrease SED	70.4	1.91 (1.16)	15.5 (12.2)
Rx23	Increase PA	47.7	1.98 (1.20)	17.9 (13.8)
(2 weeks)	Decrease SED	68.9	1.98 (1.29)	14.6 (9.7)

American adults meet these guidelines,² and developing interventions to promote physical activity is an active area of research.

Little is known about the ways in which participants modify their activity in PA interventions where the level of behavior is prescribed but not closely supervised in-person.³ Participants can increase their daily PA by increasing the number of PA bouts per day while keeping the duration of the bouts constant; they can keep the number of bouts constant but increase the duration of each bout; or participants can increase both the number of bouts per day and their duration. Evaluating the relationship between these two processes, whether they are substitutes or compliments for one another, and how they change in response to treatment can provide insights into how an intervention works and how it can be refined.

1.2 | Motivating example: The Make Better Choices study

The Make Better Choices (MBC) study was a randomized lifestyle intervention of 204 adults focused on changing PA and eating behaviors. MBC participants were randomly assigned to one of two activity-related intervention arms: (1) iPA: an increase PA arm with a goal to increase MVPA or (2) dSED: a decrease sedentary behavior arm with a goal to decrease leisure-time sedentary screen time. Detailed information about the intervention has been published elsewhere.^{4,5} Briefly, the study consisted of a 2-week baseline assessment period and a 3-week intervention period. The 3-week intervention period used a step-up approach. A participant's behavior change prescription for the first week of the intervention period (referred to as the Rx1 phase) was to attain half of their final target goal. The prescription for the last 2 weeks of the intervention period (referred to as the Rx23 phase) was to attain the full, targeted goal and to maintain that level of performance until the end of the 3-week intervention period.

PA in the MBC study was measured daily using an Actigraph accelerometer (model 7164; Actigraph, LLC, Pensacola, Florida) throughout the 5 weeks of the study. This uniaxial device measures and processes vertical acceleration as counts, providing an indication of the amount and intensity of PA.⁶ Data were recorded in 1-min epochs. Using cut-points developed by Freedson et al,⁷ MVPA was defined as an accelerometer count greater than 1951 counts/min. Following Masse et al,⁸ an exercise bout was defined as at least 10 consecutive minutes of accelerometer counts greater than 1951 counts/min with allowance for 1–2 min of counts below 1951 counts/min. Note that based on this definition, the minimum bout duration time is 8 min. See Siddique et al⁹ for further details regarding processing of the MBC accelerometer data. Compliance with wearing the accelerometer was good, with 85% of participants wearing the accelerometer for 10 h per day on 4 or more days per week throughout the study. More information on accelerometer compliance by treatment phase and study condition is provided in Section A of the supplementary materials.

Table 1 reports the percentage of days with zero bouts, mean (SD) bouts on non-zero bout days, and mean (SD) bout duration by treatment condition and study phase for the 204 participants in the MBC study. Overall, the percentage of zero-bout days tends to decrease over time in the iPA group and stays constant in the dSED group. The average number of bouts per day appears to stay the same over time and by treatment condition. Bout duration tends to increase in the iPA group.

Figures 1 and 2 in Section B of the supplementary materials display the distribution of daily number of MPVA bouts and the duration of bouts by study phase and treatment condition. A key characteristic of the data in these figures are the

skewed nature of the data. MVPA bouts are zero inflated such that the number of days with zero bouts ranges from 48% to 72% of days depending on study phase and treatment condition (Table 1). Similarly, mean duration ranges from 13 to 18 min per bout (Table 1) but some bouts last longer than an hour.

We sought to develop a model that captures these unique features of bout MVPA as measured by accelerometer: frequent measurements, zero-inflated bouts, and skewed bout durations; and allows us to estimate how bouts and their duration vary over the course of the MBC intervention and by treatment condition.

1.3 | Research overview

In this manuscript, motivated by the MBC study, we develop methods for jointly modeling both the number of exercise bouts per day and the duration of these bouts. This allows us to investigate the effect of the MBC intervention on two outcomes: number of daily PA bouts and their duration. There are several advantages to jointly modeling PA bouts and duration as opposed to fitting separate models for these outcomes. Modeling duration separately ignores days when a participant did not exercise. Bout frequency is reported every day (even if it is 0) and joint modeling allows one data type to compensate for lack of information in the other,¹⁰ potentially resulting in more precise parameter estimates. Joint modeling also allows us to explore the correlation between bout frequency and bout duration and whether, for example, changes in bout frequency and duration are negatively correlated such that participants who increase their exercise bouts reduce the duration of their bouts. Understanding these relationships between health behaviors and whether they are substitutes or compliments for one another can help inform the development of PA interventions.⁴

Originally focused on joint modeling of longitudinal and time-to-event outcomes, joint models have seen considerable development in recent years and have been extended to accommodate multiple longitudinal outcomes of different types. Most relevant to our work are joint models that model both the frequency (often zero-inflated) and intensity of a longitudinal process. Buta et al¹¹ developed a joint model of longitudinal data of drinking behavior in an alcoholism trial. Their joint model included two sub-models: a hurdle-binomial model for number of drinking days per week; and a log-normal model for the average number of drinks per drinking day in a given week. Gupta et al¹² model longitudinal blood glucose monitoring data from an observational study of pregnant women. They jointly model the number of daily glucose measurements using a Poisson model, and a linear mixed model for average daily glucose. The random effects from these two sub-models are used in a logistic regression to predict pre-term birth. Finally, Juarez-Colunga et al¹³ jointly modeled the daily number of hot flashes and a daily indicator for whether the symptoms were severe or not through the use of shared random effects.

We build on and extend these existing approaches for joint modeling of longitudinal outcomes in a number of ways in order to better understand how participants change their physical activity in an intervention study. We exploit the intensive nature of accelerometer data which is typically measured daily over long periods of time by using a complex random effects structure in order to understand the multivariate relationship between outcomes and how they change over time. We use a Poisson hurdle sub-model for daily bouts to model the effect of the intervention not only on bout frequency but also on the presence/absence of PA on a given day. And we develop and implement a mixed-effects gamma location scale model to account for the skewed, non-negative nature of the physical activity duration data and to model the effect of treatment condition and time on duration mean and duration variance.

The outline for the rest of this paper is as follows. In Section 2, we describe our joint model of physical activity bouts and their duration. In Section 3 we apply our method to the MBC study. In Section 4, we use simulation to investigate how well our model can estimate the correlation between bouts and their duration. Section 5 provides discussion and some areas for future research.

2 | METHODS

Our joint model consists of two submodels: a mixed-effects Poisson hurdle model for the number of daily MVPA bouts,¹⁴ and a mixed-effects location scale gamma regression model for bout duration.

Let n_{ij} be the total number of exercise bouts for participant i on day j such that if the participant does not engage in any MVPA bouts on day j , then $n_{ij} = 0$, otherwise $n_{ij} > 0$, where $j = 1, \dots, J_i$ and $i = 1, \dots, N$. Here, N is the total number of

participants and J_i is the total number of days that participant i wore an accelerometer. Furthermore, if $n_{ij} > 0$, then y_{ijk} is the duration (in minutes) of the k th bout for participant i on (a physically active) day j , where $k = 1, \dots, n_{ij}$. Because $y_{ijk} \geq 8$ by the definition given in Section 1.2, prior to fitting our models, we subtract 7.9 from all duration values to better satisfy the assumptions of the Gamma distribution which we use to model duration.

2.1 | Mixed-effects poisson hurdle sub-model

We assume that n_{ij} follows a Poisson hurdle distribution, which is a mixture of a point mass at zero for physically inactive days and a zero-truncated poisson distribution for physically active days:

$$Pr(n_{ij} = 0) = 1 - \pi_{ij}, \quad 0 \leq \pi_{ij} \leq 1, \quad (1)$$

$$Pr(n_{ij} = k) = \pi_{ij} \frac{\lambda_{ij}^k e^{-\lambda_{ij}}}{k!(1 - e^{-\lambda_{ij}})}, \quad k = 1, \dots, \infty, \quad 0 < \lambda_{ij} < \infty, \quad (2)$$

where $\pi_{ij} = Pr(n_{ij} > 0)$; the probability that participant i engages in at least one MVPA bout on day j . The parameter λ_{ij} in Equation (2) is the expected number of episodes on day j for the i th participant under an untruncated poisson distribution.¹⁴

We model the parameters in (1) and (2) as a function of subject-specific random effects and study covariates:

$$\text{logit}(\pi_{ij}) = \mathbf{x}_{1ij}^T \boldsymbol{\beta}_1 + \mathbf{z}_{1ij}^T \mathbf{b}_{1i} \quad (3)$$

$$\log(\lambda_{ij}) = \mathbf{x}_{2ij}^T \boldsymbol{\beta}_2 + \mathbf{z}_{2ij}^T \mathbf{b}_{2i}, \quad (4)$$

where \mathbf{x}_{1ij}^T and \mathbf{x}_{2ij}^T denote covariate vectors for the fixed effects $\boldsymbol{\beta}_1$ and $\boldsymbol{\beta}_2$, respectively, and \mathbf{z}_{1ij}^T and \mathbf{z}_{2ij}^T denote covariate vectors for the normally distributed random effects \mathbf{b}_{1i} and \mathbf{b}_{2i} , respectively.

2.2 | Mixed-effects location scale gamma sub-model

For participants who engage in at least 1 MVPA bout on day j , we assume that the duration of the k th bout on day j for the i th participant, y_{ijk} , follows a mean-parametrized gamma distribution:¹⁵

$$f(y_{ijk} | n_{ij} > 0, \mu_{ij}, \alpha_{ij}) = \frac{1}{\Gamma(\alpha_{ij})} \left(\frac{\alpha_{ij}}{\mu_{ij}} \right)^{\alpha_{ij}} y_{ijk}^{\alpha_{ij}-1} \exp\left(-\frac{y_{ijk} \alpha_{ij}}{\mu_{ij}}\right) \text{ for } y_{ijk} > 0, \quad (5)$$

where $\Gamma(\cdot)$ denotes the gamma function, μ_{ij} ($\mu_{ij} > 0$) is the expected duration of a bout on day j for participant i , and $\frac{\mu_{ij}^2}{\alpha_{ij}}$ is its variance. As mentioned above, to better satisfy the assumptions of the gamma distribution, we subtract 7.9 from all duration values. Note that the shape parameter, α_{ij} ($\alpha_{ij} > 0$) is—like the mean parameter—indexed by participant and day.

As in (3) and (4) we model the mean and shape parameters in (5) as a function of subject-specific random effects and study covariates via the following log-linear models:

$$\log(\mu_{ij}) = \mathbf{x}_{3ij}^T \boldsymbol{\beta}_3 + \mathbf{z}_{3ij}^T \mathbf{b}_{3i} \quad (6)$$

$$\log(\alpha_{ij}) = \mathbf{x}_{4ij}^T \boldsymbol{\beta}_4 + \mathbf{z}_{4ij}^T \mathbf{b}_{4i}, \quad (7)$$

where \mathbf{x}_{3ij}^T and \mathbf{x}_{4ij}^T denote covariate vectors for the fixed effects $\boldsymbol{\beta}_3$ and $\boldsymbol{\beta}_4$, respectively, and \mathbf{z}_{3ij}^T and \mathbf{z}_{4ij}^T are covariate vectors for the random effects \mathbf{b}_{3i} and \mathbf{b}_{4i} , respectively.

We refer to the model in (5)–(7) as a mixed-effects location scale gamma regression model as it closely follows the approach outlined by Hedeker et al.¹⁶ for modeling both the location and scale of longitudinal data as a function of fixed and random effects.

2.3 | Joint poisson hurdle gamma model

The joint poisson hurdle gamma model is obtained by connecting the four models (two sub-models) in (3)–(4), and (6)–(7). Together, these four models provide a rich description of PA behavior in the MBC study and how it changes over time and by treatment condition. Model (3) allows us to test whether the MBC intervention increases the probability that a participant will exercise on a given day. Model (4) allows us to test whether the intervention increases the frequency of bouts on days when a participant exercises. Model (6) models the duration of a participant's PA bouts and whether duration increases over time and/or by treatment condition. Finally, model (7) characterizes changes in a participant's bout duration variability over time and by treatment condition. Importantly, connecting these four models through the use of random effects allows us to estimate the correlations of these outcomes and their changes over time. These quantities can help inform researchers when designing physical activity interventions.

Let $\beta = (\beta_1, \beta_2, \beta_3, \beta_4)$ denote the vector of fixed effects in (3)–(4) and (6)–(7) and let $\mathbf{b}_i = (\mathbf{b}_{1i}, \mathbf{b}_{2i}, \mathbf{b}_{3i}, \mathbf{b}_{4i})$ denote the random effects which we assume are independently and identically distributed from a multivariate normal distribution (dimension p) with mean zero and an unstructured covariance matrix Σ . We assume conditional independence—that given the random effects, n_{ij} and y_{ijk} are independent as are repeated measurements of n_{ij} and y_{ijk} within a participant. Let N_i summarize the n_{ij} values for participant i and Y_i summarize their duration values y_{ijk} . The likelihood for the i th participant is then:

$$\begin{aligned} L_i(\beta, \Sigma) &= f(N_i, Y_i | \beta, \Sigma) = \int_{\mathbf{b}_i} f(N_i, Y_i | \beta, \mathbf{b}_i, \Sigma) f(\mathbf{b}_i | \Sigma) d\mathbf{b}_i \\ &= \int_{\mathbf{b}_i} \prod_{j=1}^{N_i} (1 - \pi_{ij})^{1-d_{ij}} \left\{ \frac{\pi_{ij} \lambda_{ij}^{n_{ij}} \exp(-\lambda_{ij})}{n_{ij}! \{1 - \exp(-\lambda_{ij})\}} \prod_{k=1}^{n_{ij}} \frac{1}{\Gamma(\alpha_{ij})} \left(\frac{\alpha_{ij}}{\mu_{ij}} \right)^{\alpha_{ij}} y_{ijk}^{\alpha_{ij}-1} \exp\left(-\frac{y_{ijk} \alpha_{ij}}{\mu_{ij}}\right) \right\}^{d_{ij}} \\ &\quad \times \left(\frac{1}{2\pi} \right)^{\frac{p}{2}} |\Sigma|^{-\frac{1}{2}} \exp \left\{ -\frac{1}{2} (\mathbf{b}_i' \Sigma^{-1} \mathbf{b}_i) \right\} d\mathbf{b}_i, \end{aligned} \quad (8)$$

where d_{ij} is an indicator that $n_{ij} > 0$ and π_{ij} , λ_{ij} , α_{ij} , and μ_{ij} are defined above.

2.4 | Daily minutes of physical activity

A benefit of our model is its ability to decompose physical activity into its components: number of bouts and the duration of each bout and to estimate the effects of covariates on these outcomes. However, PA recommendations are made at the day or week level and researchers may wish to understand the effects of treatment on total daily duration both overall and on exercise days.

The total PA minutes for participant i on day j is the sum of the duration of their n_{ij} bouts. That is, $\sum_{k=1}^{n_{ij}} y_{ijk} + 7.9 = n_{ij} \bar{y}_{ij} + 7.9$ where \bar{y}_{ij} is the average bout duration for participant i on day j .

The mean of the Poisson hurdle distribution in terms of the parameters in (1) and (2) is

$$E(n_{ij} | \pi_{ij}, \lambda_{ij}) = \pi_{ij} \frac{\lambda_{ij}}{1 - \exp(-\lambda_{ij})}, \quad (9)$$

where π_{ij} is the probability that $n_{ij} > 0$. For mean duration, since the model in (5) does not depend on covariates measured at the bout-level, $E(y_{ijk}) = E(\bar{y}_{ij}) = \mu_{ij}$.

Due to conditional independence of n_{ij} and y_{ijk} , the expectation of $n_{ij} \times \bar{y}_{ij}$ can be written as a product of their expectations, that is:

$$E(n_{ij} \times \bar{y}_{ij} | \pi_{ij}, \lambda_{ij}, \mu_{ij}) = E(n_{ij} | \pi_{ij}, \lambda_{ij}) E(\bar{y}_{ij} | \mu_{ij}) = \frac{\pi_{ij} \lambda_{ij} \mu_{ij}}{1 - \exp(-\lambda_{ij})}. \quad (10)$$

Plugging in (3), (4), and (6) we obtain

$$E(n_{ij} \times y_{ijk} | \mathbf{b}_i) = \frac{\exp(\mathbf{x}_{2ij}^T \beta_2 + \mathbf{z}_{2ij}^T \mathbf{b}_{2i} + \mathbf{x}_{3ij}^T \beta_3 + \mathbf{z}_{3ij}^T \mathbf{b}_{3i})}{\left[1 + \exp(-\mathbf{x}_{1ij}^T \beta_1 - \mathbf{z}_{1ij}^T \mathbf{b}_{1i}) \right] \left[1 - \exp(-\exp(\mathbf{x}_{2ij}^T \beta_2 + \mathbf{z}_{2ij}^T \mathbf{b}_{2i})) \right]}. \quad (11)$$

The constant 7.9 is added to the quantities in (10) and (11) to put total duration back on its original scale. In Section C of the supplementary materials, we provide details on integrating out the random effects in (11) in order to calculate marginal daily duration by treatment group and time. If the goal is to calculate daily minutes of PA on exercise days, (9) is replaced with the mean of a *truncated* Poisson distribution $E(n_{ij} \mid n_{ij} > 0, \lambda_{ij})$.

3 | APPLICATION TO THE MBC STUDY

We modeled the MBC data using the joint model described above with and without modeling the shape parameter in (7). We used several different models with different random effect structures. The simplest model consisted of random intercepts only in each sub-model and no model for the shape parameter. The most complex model included random intercept, Rx1, and Rx23 terms in all four sub-models. In all of our models, the fixed-effects covariate vectors were the same with main effects for study phase and study phase by treatment interaction terms and we did not include any bout-specific covariates (eg, time of day or weather) in our models so that $\mathbf{x}_{1ij} = \mathbf{x}_{2ij} = \mathbf{x}_{3ijk} = \mathbf{x}_{4ijk} = \mathbf{x}_{ij}$ where

$$\mathbf{x}_{ij}^T = (1, \text{Rx1}_{ij}, \text{Rx23}_{ij}, \text{Rx1}_{ij} * \text{iPA}_i, \text{Rx23}_{ij} * \text{iPA}_i). \quad (12)$$

In (12), Rx1_{ij} and Rx23_{ij} are indicator variables for study phase, and $\text{Rx1}_{ij} * \text{iPA}_i$ and $\text{Rx23}_{ij} * \text{iPA}_i$ are treatment by study phase interaction terms. The regression coefficients for these two interaction terms are our estimates of interest, the difference between iPA and dSED at follow-up for the outcome in the sub-model. For example, in the sub-model in (1), the regression coefficient on the Rx23 by iPA interaction term is the difference in log odds at Rx23 of participant i engaging in any bouts on day j in the iPA group versus the dSED group. Note that (12) does not include a main effect for treatment under the assumption that the distribution of responses at baseline can be assumed to be equal in a randomized trial.¹⁷

3.1 | Parameter estimation, model selection, model fit

We used a Bayesian approach to obtain the posterior distribution of our model parameters. We specified non-informative normal priors with mean 0 and variance 1,000 for the regression coefficients, and a $\text{IW}(\mathbf{I}_k, k + 1)$ inverse-Wishart prior¹⁸ for the covariance matrix Σ_k of the random effects where k is the dimension of the combined collection of random effects in each of the four sub-models and \mathbf{I}_k is a $k \times k$ identity matrix. Based on the above priors and the likelihood in (8), the posterior distribution of the fixed effects β , random effects \mathbf{b} and their covariance Σ is

$$p(\beta, \mathbf{b}, \Sigma \mid N, Y) \propto \prod_{i=1}^N f(N_i, Y_i \mid \beta, \mathbf{b}_i, \Sigma) p(\mathbf{b}_i \mid \Sigma) \pi(\Sigma) \pi(\beta). \quad (13)$$

Markov Chain Monte Carlo (MCMC) using JAGS¹⁹ via the R package runjags²⁰ was used to obtain draws from the joint posterior distribution in (13). The number of iterations needed to achieve convergence depended on the number of random effects in our models. Convergence of the MCMC chains was monitored using trace plots, density plots, and Gelman-Rubin statistics.²¹ JAGS code for our model is included in Section D of the supplementary materials.

As recommended by Gelman et al²² for use with hierarchical Bayesian models where the number of parameters increases with the sample size, we used the Widely Applicable Information Criterion (WAIC) to select among models with different random effects. WAIC is based on the log pointwise predictive density (lppd) which—in our application—is the sum of each participant's log likelihood averaged over the posterior distribution of the model parameters and can be calculated using MCMC draws. Let $[\beta^s, \mathbf{b}_i^s, \Sigma^s]$, $s = 1, \dots, S$ be draws from the posterior distribution in (13) and $p(N_i, Y_i \mid \beta^s, \mathbf{b}_i^s, \Sigma^s)$ be the likelihood for participant i based on the s th draw. The lppd is defined as

$$\text{lppd} = \sum_{i=1}^N \log \left(\frac{1}{S} \sum_{s=1}^S p(N_i, Y_i \mid \beta^s, \mathbf{b}_i^s, \Sigma^s) \right). \quad (14)$$

To adjust for overfitting, the WAIC includes a correction to the lppd for the effective number of parameters:

$$p_w = \sum_i^N V_{s=1}^S (\log p(N_i, Y_i \mid \beta^s, \mathbf{b}_i^s, \Sigma^s)), \quad (15)$$

where $V_{s=1}^S$ represents the sample variance. To put it on a deviance scale, WAIC is defined as $-2\text{lppd} + 2p_w$.

After identifying the best fitting model using the WAIC, we evaluated how well our final model fit the observed data using posterior predictive checking.²³ For each draw from the posterior distribution of our parameters we generated replicated values n_{ij}^{rep} and y_{ijk}^{rep} corresponding to daily number of bouts and their duration. We used four different test statistics to capture how well the replicated data resembled the observed data. These test statistics are based on the observed quantities reported in Table 1: the proportion of days with zero bouts, the mean number of non-zero bouts, mean bout duration, and the standard deviation of bout duration. We calculated these test statistics at each treatment phase and compared them to their values based on the replicated data both in terms of the magnitude of the difference as well as by calculating posterior predictive probabilities (p_B), the probability that the replicated data is more extreme than the observed data.

We also generated QQ plots to assess how well the Poisson hurdle and Gamma regression models fit our data. We calculated quantiles using n_{ij}^{rep} and y_{ijk}^{rep} and plotted them against their observed quantiles. For the QQ plots we used 10 draws from the posterior distribution to generate 10 QQ plots for both number of bouts and their duration.

3.2 | Results

Table 2 in Section E of the supplementary materials reports the WAIC for each of the models with varying numbers of random effects. Despite containing a random effects variance-covariance matrix with 78 parameters, the model with random intercepts, Rx1, and Rx23 in all four sub-models had the lowest WAIC. Further, modeling the duration shape

TABLE 2 Posterior estimates from the joint mixed-effects Poisson gamma mixed-effects location scale model with random effects for intercept, Rx1, and Rx23.

Variable	Mean	Lower 95% CI	Upper 95% CI
<i>Logistic regression on probability of exercise day</i>			
Intercept	−0.987	−1.172	−0.800
Rx1	−0.081	−0.349	0.185
Rx23	−0.046	−0.300	0.207
PA*Rx1	1.090	0.728	1.451
PA*Rx23	1.031	0.682	1.377
<i>Loglinear regression on mean number of bouts</i>			
Intercept	−0.045	−0.187	0.090
Rx1	−0.034	−0.277	0.203
Rx23	−0.039	−0.249	0.163
PA*Rx1	0.090	−0.145	0.323
PA*Rx23	0.160	−0.039	0.352
<i>Loglinear regression on duration mean</i>			
Intercept	1.540	1.401	1.686
Rx1	0.070	−0.160	0.294
Rx23	−0.061	−0.272	0.144
PA*Rx1	0.413	0.176	0.645
PA*Rx23	0.639	0.430	0.851
<i>Loglinear regression on duration shape</i>			
Intercept	−0.432	−0.536	−0.327
Rx1	0.056	−0.145	0.268
Rx23	−0.074	−0.230	0.083
PA*Rx1	0.139	−0.068	0.343
PA*Rx23	0.020	−0.127	0.171

TABLE 3 Posterior predictive checks from the joint mixed-effects Poisson gamma location-scale model with random effects for intercept, Rx1, and Rx23 applied to the MBC data.

Test statistic	Baseline			Rx1			Rx23		
	Obs	Rep	ppp	Obs	Rep	ppp	Obs	Rep	ppp
Prop. days any bouts	0.304	0.304	0.96	0.392	0.392	1.00	0.410	0.410	0.95
Mean daily bouts	0.550	0.551	0.99	0.741	0.739	0.97	0.812	0.809	0.88
Mean bout duration	13.4	13.4	0.96	16.2	16.2	0.89	16.6	16.6	0.95
Bout duration SD	9.07	9.16	0.95	11.2	12.7	0.26	12.4	14.1	0.13

Note: Four test statistics were used to assess model fit at each of the three study time points: proportion of days with any bouts, mean number of daily bouts, mean bout duration, standard deviation of bout duration.

Abbreviations: Obs, observed value; ppp, posterior predictive probability; Rep, mean replicated value.

parameter, that is, modeling both duration mean and variance demonstrated an improvement in model fit, resulting in a reduction in WAIC of 102.4 compared to a model where duration shape is assumed to be constant.

Table 2 reports the posterior means and 95% credible intervals from this best fitting model for both fixed effects and variances from the random effects variance-covariance matrix. In all four models, the main effects for Rx1 and Rx23 are not significant indicating no change over time in the dSED group on all four outcomes. Focusing our attention on the time by treatment interaction terms, we see significant effects in the Poisson hurdle component and the mean model for duration. These results suggest that MBC iPA participants increased their physical activity by having more exercise days and also by increasing the duration of their exercise bouts; not by increasing the number of bouts on days they exercised. Interestingly, the treatment effects in the shape model were not significant. Since the mean-parameterized gamma distribution with mean μ and shape α has variance equal to μ^2/α , the parameters of the shape sub-model cannot be interpreted directly since duration variance is a function of both the parameters in (6) and (7) (Supplementary materials Section F). Still, these results suggest that increases in duration variability were due to changes in mean duration rather than a change in the shape parameter.

Table 3 reports the results from posterior predictive checking of the fit of our final model. Overall, our model shows adequate fit. None of the four test statistics at the three time points has a two-sided posterior predictive probability less than 0.10.

Figures 3 and 4 in Section G of the supplementary materials are QQ plots of posterior predictive replicates of the daily bout values and duration values versus their observed values, respectively. In both plots, the distribution of the replicated data are similar to that of the observed data.

Although our interest is on the effect of treatment on all four outcomes in the joint model, it is possible to perform a global test of significance of the treatment effects, to test whether *any* of the time by treatment interactions are significant. To do this, we calculated the proportion of MCMC draws where the treatment effects in the hurdle, truncated Poisson, and gamma mean models were greater than zero or the treatment effect in the gamma shape model was less than zero. At both Rx1 and Rx23, the predictive probability for this global test was $< .0001$.

Table 4 in Section H of the supplementary materials reports the lower diagonal of the 12×12 random effects correlation matrix from the final model. Correlations whose 95% credible intervals did not include zero are noted in bold. The correlation between the random intercept for the hurdle sub-model and the random intercept for the number of bouts sub-model was 0.70 indicating that participants who had a greater than average probability of exercising also engaged in more bouts per day than average. The correlation of the random Rx23 effect between these two sub-models was also significant, such that those participants with greater than average increases in their probability of exercising at Rx23 also had greater than average increases in bouts per day ($\hat{\rho} = 0.55$).

Within all four sub-models, the correlations between the random effects for Rx1 and Rx23 were large and significant. For example, participants who had greater than average changes in their probability of exercising at Rx1 also had greater than average changes in their probability of exercising at Rx23 ($\hat{\rho} = 0.80$). However, only in the shape model were random intercepts correlated with random effects at Rx1 and Rx23. Here, participants with lower than average shape parameter values at baseline tended to have higher than average changes in shape values at follow up ($\hat{\rho} = -0.33$ and -0.39 , respectively).

It is also worth noting that while there were significant correlations between random effects associated with the Poisson sub-models and significant correlations between random effects associated with the gamma sub-model, almost

none of the correlations of the random effects across these two sub-models were significant. For example, the correlation between the random intercept of the number of bouts model and the random intercept for the duration model (a quantity of particular interest to interventionists) was only 0.06. These low correlations may explain why—as described below—there is minimal loss in precision for this particular data set when fitting separate models for bouts per day and bout duration.

3.3 | Separate models for bouts and duration

The primary benefit of our joint modeling approach is the ability to estimate the correlation between the change in exercise bouts and their duration as well as the ability to perform a global test of treatment. Still, both joint and separate models allow one to calculate treatment effects. Therefore, we fit separate models of exercise bouts and their duration in order to compare these models to our joint modeling approach. The results from these separate models are reported in Section I of the supplementary materials. A comparison of treatment effects from the joint model and the separate models is displayed in Supplementary Materials Figure 5. Overall, there is very little difference in treatment effects between joint and separate models, both in terms of their point estimates and the width of their credible intervals.

3.4 | Daily duration

Table 4 reports daily duration—on all days as well as on exercise days—by treatment condition and time. Results are provided based on a joint model as well as based on separate modeling of bouts and duration. The conclusions are similar to those in Table 2. Daily duration does not change over time in the dSED group but does change in the iPA group such that by Rx23, iPA participants engage in 11 more minutes of MVPA per day compared to dSED participants.

TABLE 4 Daily minutes of physical activity by treatment condition and time.

Group	Joint model					Separate models				
	Mean	SE	LCI	UCI	Width	Mean	SE	LCI	UCI	Width
<i>All days</i>										
Baseline	7.85	0.64	6.73	9.19	2.46	7.85	0.62	6.73	9.05	2.32
iPA Rx1	17.85	1.88	14.55	21.93	7.38	17.32	1.67	14.27	20.82	6.55
dSED Rx1	8.97	1.09	7.05	11.24	4.19	8.84	1.05	7.07	11.12	4.05
Rx1 Diff	8.89	1.85	5.42	12.61	7.19	8.48	1.75	5.23	12.04	6.81
iPA Rx23	20.05	2.12	16.19	24.81	8.62	19.27	1.86	16.03	23.33	7.30
dSED Rx23	8.97	1.03	7.16	11.16	4.00	8.70	0.93	7.07	10.64	3.57
Rx23 Diff	11.08	2.06	7.38	15.53	8.15	10.57	1.83	7.13	14.26	7.13
<i>Exercise days</i>										
Baseline	22.66	0.99	20.72	24.64	3.92	22.66	0.92	20.93	24.53	3.60
iPA Rx1	31.03	2.13	27.22	35.56	8.34	31.58	2.13	27.89	36.21	8.32
dSED Rx1	24.19	1.59	21.43	27.56	6.13	24.85	1.66	21.91	28.46	6.55
Rx1 Diff	6.84	2.46	2.47	12.02	9.55	6.73	2.47	2.07	11.48	9.41
iPA Rx23	34.08	2.26	30.06	38.77	8.71	34.51	2.22	30.53	39.10	8.57
dSED Rx23	22.89	1.25	20.64	25.41	4.77	23.26	1.25	20.96	25.89	4.93
Rx23 Diff	11.19	2.38	6.75	15.89	9.14	11.24	2.31	6.97	16.03	9.06

Note: The top half of the table averages over all days (including days with zero bouts). The bottom half of the table only includes days with one or more bouts (exercise days). Results are provided based on a joint model of bouts and their duration and also based on fitting two separate models for bouts and their duration. Abbreviations: dSED, decrease sedentary group; iPA, increase PA group.

Interestingly, while point estimates are similar between results based on a joint model and those based on separate models, the standard errors are smaller for separate models as compared to joint models.

4 | SIMULATION STUDY

As our primary motivation for using a joint model is to estimate the correlation between bouts and their duration, we performed a simulation study using a factorial design to evaluate the ability of our joint model to accurately estimate the correlations between these two processes.

We simulated number of daily bouts (n_{ij}) and their duration (y_{ijk}) using the joint Poisson hurdle gamma model described in Section 2. The parameters of the distributions were based on the following random intercept mixed-effects models:

$$\text{logit}(\pi_{ij}) = \alpha_0 + \alpha_1(\text{time}_{ij} \times \text{trt}_i) + b_{1i} \quad (16)$$

$$\log(\lambda_{ij}) = \beta_0 + \beta_1(\text{time}_{ij} \times \text{trt}_i) + b_{2i} \quad (17)$$

$$\log(\mu_{ij}) = \gamma_0 + \gamma_1(\text{time}_{ij} \times \text{trt}_i) + b_{3i} \quad (18)$$

$$\log(\alpha_{ij}) = \phi_0 + \phi_1(\text{time}_{ij} \times \text{trt}_i) + b_{4i}, \quad (19)$$

where $\text{time}_{ij} \times \text{trt}_i$ is the time by treatment interaction and the random effects \mathbf{b}_i follow a multivariate normal distribution with mean 0 and variance-covariance matrix Σ where

$$\Sigma = \begin{pmatrix} 1 & & & \\ 0.7 & 1 & & \\ \rho_{13} & \rho_{23} & 1 & \\ \rho_{14} & \rho_{24} & -0.2 & 1 \end{pmatrix}. \quad (20)$$

Since the variances in Σ are equal to 1, its off-diagonal terms are correlations. In a simplified version of the MBC data, we assumed $N = 200$ participants with half of participants belonging to the treatment condition, half to the control condition. Each participant was measured for 7 days at baseline ($\text{time}_{ij} = 0$) and 7 days at follow-up ($\text{time}_{ij} = 1$). Across all simulations, based on values from the MBC study, we fixed α_0 in (16) to 1.1 corresponding to 25% zero bout days and set the coefficients α_1 , and β_1 in (16) and (17) to $\log(3)$. The coefficient β_0 in (17) was set to $\log(2)$. The parameters in the duration models in (18) and (19) were fixed at $\gamma_0 = 1.5$, $\gamma_1 = 0.5$, $\phi_0 = -0.4$, and $\phi_1 = 0.14$.

Only the correlations in (20) varied across simulation scenarios. The parameters ρ_{13} , ρ_{14} , ρ_{23} , ρ_{24} , were set to either 0.2 or 0.5. These were deemed to be plausible values and would allow us to evaluate how well our model is able to estimate these correlations when their true values are both small and moderate.

With four factors set to two levels, there are a total of $2^4 = 16$ scenarios. For each scenario, we generated 100 simulated data sets and fit a joint model to each data set. Our targets of inference were the four correlation parameters in (20) which we evaluated in terms of the following performance criteria: bias, variance, mean squared error (MSE), coverage of the 95% credible interval, and width of the credible interval.

4.1 | Simulation results

Table 5 presents the simulation results for the four correlation coefficients that varied across scenarios. Each row is based on 800 simulations that average over the levels of the three other correlation coefficients. All correlation coefficients were estimated with low bias, variance and MSE. Coverage tended to be slightly below the nominal level and the width of the 95% credible interval was narrower when the true value of the correlation parameter was 0.5 versus 0.2.

TABLE 5 Bias, variance, mean squared error (MSE), coverage, and average width of the 95% credible interval of parameter estimates from the simulation study.

Parameter	True value	Bias	Variance	MSE	Coverage	95% CI width
ρ_{13}	0.2	0.011	0.009	0.01	0.922	0.35
	0.5	-0.005	0.006	0.01	0.930	0.29
ρ_{14}	0.2	-0.001	0.007	0.01	0.934	0.33
	0.5	-0.009	0.005	0.01	0.936	0.28
ρ_{23}	0.2	-0.005	0.005	0.01	0.924	0.29
	0.5	-0.004	0.003	0.00	0.948	0.23
ρ_{24}	0.2	-0.006	0.005	0.00	0.939	0.28
	0.5	-0.009	0.003	0.00	0.935	0.22

Note: All correlation coefficients were estimated with low bias, variance and MSE. Coverage tended to be slightly below the nominal level and the width of the 95% credible interval was narrower when the true value of the correlation parameter was 0.5 versus 0.2.

5 | DISCUSSION

We have described an approach for modeling physical activity data from a lifestyle intervention trial that jointly models daily exercise bouts and the duration of those bouts. We extended existing approaches for joint modeling by using sub-models that accommodate the unique distributional properties of our outcomes—zero inflated bout frequencies and skewed bout durations. In addition, in our model for PA duration, we model both the mean and shape parameters of the gamma distribution as a function of fixed and random effects, the first time that we are aware of that this has been done for a gamma-distributed longitudinal outcome.

In our analysis of the MBC data, we found significant treatment effects on the number of non-zero exercise days as well as on duration mean. Participants in the iPA condition increased their MVPA compared to the dSED condition by exercising on more days and by increasing their bout duration. They did not, however, increase the number of bouts per day, and participants in both conditions averaged a little less than two bouts per day throughout the study on days they engaged in any exercise. Participants in the iPA condition also increased their bout duration variability over time.

There are numerous advantages to our joint modeling approach as compared to modeling bout frequency and its duration in separate models. The joint model allows researchers to estimate the correlation between these outcomes which is an important quantity of interest to behavioral scientists. If change in these outcomes is uncorrelated, as was seen in the MBC data, then researchers can focus on either outcome in order to increase the volume of physical activity. On the other hand, if the change in bouts and their duration is negatively correlated, then interventionists may need target both outcomes with the understanding that increasing one outcome, say number of bouts per day, may decrease the duration of each individual bout. In an intervention study, a joint model also allows one to perform a global test of whether the intervention has an effect on any outcome of interest including outcomes across sub-models.

Additional covariates can be incorporated into our model and not all sub-models need to use the same covariates. Day-level covariates like weather and weekend/weekday can be included into the bout sub-model,²⁴ and both day-level and bout-level predictors like time of day can be incorporated into the duration model. Including these additional variables into the models can help shed light on what contextual factors may play a role in a person's decision to engage in physical activity and whether these covariates moderate the effect of treatment. In settings where there are multiple bouts per day, a three-level model with a day-level random effect can be incorporated in the the duration sub-model to capture day-level variability. If compliance with wearing the accelerometer is an issue, an additional sub-model could be added indicating whether the accelerometer was worn on a given day.

It is noteworthy that in our analysis of the MBC data we found little difference in estimates of treatment effects from our joint model as compared to estimates using separate models for bouts and duration. Based on work by Su et al,²⁵ this is to be expected since the benefits of joint modeling in longitudinal data are reflected in more accurate estimates of intercepts, rather than treatment effects. If the distribution of random effects differed by treatment condition—as may occur in an observational study where “treatment” is not randomly assigned—differences in estimates of treatment effects

between the joint and separate modeling approaches may arise. In settings where there are a fewer number of days, a joint modeling approach may provide gains in efficiency.

In our estimates of daily minutes of physical activity, we found that point estimates were similar between estimates based on a joint model and those based on separate models. However, standard errors were smaller for estimates based on separate models. The reason for this discrepancy is not clear and future work will attempt to elucidate these differences in results.

Our model can be used to identify different features of physical activity and its change over time in terms of frequency of exercise days, number of bouts per day, bout duration, and bout duration variance. Examples of these “behavioral phenotypes” are shown in Figures 6 and 7 in Section J of the supplementary materials. Behavioral phenotypes that are measured during the intervention period may be good predictors of distal outcomes like weight gain or behavior maintenance.²⁶ If particular behavioral phenotypes (eg, composed of distinct combinations of bout duration, bout duration variability, frequency of days with exercise) appear to be associated with distal outcomes of clinical interest, then this would support tailoring intervention recommendations to include specific goals related to the components that are characteristic of the optimal behavioral phenotype. We are interested in incorporating these distal outcomes into our joint model where features from the intervention period are used as predictors.

Other work will involve relaxing the normality assumption of the distribution of random effects to allow for non-linear correlations in addition to modeling these correlations as a function of participant characteristics to better understand how behaviors change over the course of an intervention.²⁷⁻²⁹ Identifying behaviors that change together (ie, are positively correlated) or behaviors that are substitutes for one another (ie, are negatively correlated) is of particular interest to researchers when designing interventions that target multiple behaviors.

While the physical activity guidelines are expressed in minutes per week, physical activity is in fact a multidimensional construct that occurs at the day or even more granular levels. Our model allows researchers to decompose physical activity into its many parts: (1) its occurrence; (2) its frequency; (3) its duration; and (4) its variability. Understanding these different components, how they covary, and how they change over time will allow researchers to better understand the effects of their interventions, help design new and better interventions, and identify those features that lead to durable behavior change.

ACKNOWLEDGEMENTS

Siddique and Daniels were partially supported by NIH grant R01 HL158963. Siddique, Battalio, Spring, and Hedeker were supported by NIH grant R01 DK125414. The MBC study was supported by NIH grant R01 HL075451.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Juned Siddique  <https://orcid.org/0000-0002-1501-4152>

Gül Inan  <https://orcid.org/0000-0002-3981-9211>

REFERENCES

1. U.S. Department of Health and Human Services. *Physical Activity Guidelines for Americans*. Washington, DC: U.S. Department of Health and Human Services; 2008:2008.
2. Carlson SA, Fulton JE, Schoenborn CA, Loustalot F. Trend and prevalence estimates based on the 2008 physical activity guidelines for Americans. *Am J Prev Med*. 2010;39(4):305-313.
3. Conroy DE, Hedeker D, McFadden HG, et al. Lifestyle intervention effects on the frequency and duration of daily moderate-vigorous physical activity and leisure screen time. *Health Psychol*. 2017;36(4):299.
4. Spring B, Schneider K, McFadden H, et al. Make better choices (MBC): study design of a randomized controlled trial testing optimal technology-supported change in multiple diet and physical activity risk behaviors. *BMC Public Health*. 2010;10(1):586.
5. Spring B, Schneider K, McFadden HG, et al. Multiple behavior changes in diet and activity: a randomized controlled trial using mobile technology. *Arch Intern Med*. 2012;172(10):789-796.
6. John D, Freedson P. Actigraph and actical physical activity monitors: a peek under the hood. *Med Sci Sports Exerc*. 2012;44(1):S86.
7. Freedson PS, Melanson E, Sirard J. Calibration of the computer science and applications, Inc. accelerometer. *Med Sci Sports Exerc*. 1998;30:777-781.
8. Mâsse LC, Fuemmeler BF, Anderson CB, et al. Accelerometer data reduction: a comparison of four reduction algorithms on select outcome variables. *Med Sci Sports Exerc*. 2005;37:S544-S554.

9. Siddique J, De Chavez PJ, Craft LL, Freedson P, Spring B. The effect of changes in physical activity on sedentary behavior: results from a randomized lifestyle intervention trial. *Am J Health Promot.* 2017;31(4):287-295.
10. Hatfield LA, Hodges JS, Carlin BP. Joint models: when are treatment estimates improved? *Stat Interface.* 2014;7(4):439-453.
11. Buta E, O'Malley SS, Gueorguieva R. Bayesian joint modelling of longitudinal data on abstinence, frequency and intensity of drinking in alcoholism trials. *J R Stat Soc A Stat Soc.* 2018;181(3):869-888.
12. Gupta R, Khoury JC, Altaye M, Jandarov R, Szczesniak RD. Flexible multivariate joint model of longitudinal intensity and binary process for medical monitoring of frequently collected data. *Stat Med.* 2021;40(7):1845-1858.
13. Juarez-Colunga E, Silva GL, Dean CB. Joint modeling of zero-inflated panel count and severity outcomes. *Biometrics.* 2017;73(4):1413-1423.
14. Neelon BH, O'Malley AJ, Normand SLT. A Bayesian model for repeated measures zero-inflated count data with application to outpatient psychiatric service use. *Stat Model.* 2010;10(4):421-439.
15. Rodrigues-Motta M, Galvis Soto DM, Lachos VH, et al. A mixed-effect model for positive responses augmented by zeros. *Stat Med.* 2015;34(10):1761-1778.
16. Hedeker D, Mermelstein RJ, Demirtas H. An application of a mixed-effects location scale model for analysis of ecological momentary assessment (EMA) data. *Biometrics.* 2008;64(2):627-634.
17. Fitzmaurice GM, Laird NM, Ware JH. *Applied Longitudinal Analysis.* Hoboken, NJ: John Wiley & Sons; 2012.
18. Gelman A, Hill J. *Data Analysis Using Regression and Multilevel/Hierarchical Models.* New York, NY: Cambridge University Press; 2006.
19. Plummer M. JAGS Version 4.3.0 user manual. 2017.
20. Denwood MJ. Runjags: an R package providing Interface utilities, model templates, parallel computing methods and additional distributions for MCMC models in JAGS. *J Stat Softw.* 2016;71(9):1-25.
21. Gelman A, Rubin DB. Inference from iterative simulation using multiple sequences. *Stat Sci.* 1992;7(4):457-472.
22. Gelman A, Hwang J, Vehtari A. Understanding predictive information criteria for Bayesian models. *Stat Comput.* 2014;24(6):997-1016.
23. Gelman A, Carlin JB, Stern HS, Dunson DB, Vehtari A, Rubin DB. *Bayesian Data Analysis.* Boca Raton, FL: CRC Press; 2013.
24. Welch WA, Spring B, Phillips SM, Siddique J. Moderating effects of weather-related factors on a physical activity intervention. *Am J Prev Med.* 2018;54:e83-e89.
25. Su L, Tom BD, Farewell VT. Bias in 2-part mixed models for longitudinal semicontinuous data. *Biostatistics.* 2009;10(2):374-389.
26. Spring B, Stump TK, Battalio SL, et al. Digitally characterizing the dynamics of multiple health behavior change. *Health Psychol.* 2021;40(12):897-908.
27. Daniels MJ, Zhao YD. Modelling the random effects covariance matrix in longitudinal data. *Stat Med.* 2003;22(10):1631-1647.
28. Zhu H, Luo S, DeSantis SM. Zero-inflated count models for longitudinal measurements with heterogeneous random effects. *Stat Methods Med Res.* 2017;26(4):1774-1786.
29. Pugach O, Hedeker D, Mermelstein R. A bivariate mixed-effects location-scale model with application to ecological momentary assessment (EMA) data. *Health Serv Outcomes Res Methodol.* 2014;14(4):194-212.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Siddique J, Daniels MJ, Inan G, Battalio S, Spring B, Hedeker D. Joint modeling the frequency and duration of accelerometer-measured physical activity from a lifestyle intervention trial. *Statistics in Medicine.* 2023;42(28):5100-5112. doi: 10.1002/sim.9903