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Activity pacing in daily life: A within-day analysis

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Introduction

Activity pacing is a central concept underlying chronic pain theory and treatment, yet it is not well-characterized. It has been defined broadly as, "...regulation of activity level and/or rate in the service of an adaptive goal or goals" [p. 465, 34]. The two most common pacing domains examined in pain research are (1) slowing down/moving slowly and (2) breaking up activities into smaller pieces [35].

Pacing skills are often taught in pain treatment. We refer to this type of pacing as *programmatically pacing*. The specific goals of this training vary depending on the theoretical orientation of the treatment and include pain reduction, energy conservation (or reduced fatigue), and/or increased overall productivity. The two theoretical models guiding pacing treatment are: Operant Theory (OPT) and Energy Conservation (EC) [34]. OPT emphasizes that all behavior, including pacing, is maintained by reinforcement (i.e., the "pay-off" of the behavior) [14], such as reduced pain or increased productivity [15]. OPT-based interventions teach adaptive pacing behaviors that aim to limit the extent to which activity is symptom-contingent (e.g., reduce excessive resting when pain or fatigue are high) in order to achieve pre-determined activity goals [14]. EC-based interventions, on the other hand, seek to preserve energy for completing valued activities [16] while reducing overall pain and fatigue [34]. The existence of these two different conceptual traditions and definitions of adaptive pacing likely contributes to the current lack of clarity about the nature and impact of pacing.

Another source of confusion is limited knowledge about the pacing behaviors people enact in daily life without pacing instruction, or *naturalistic pacing* [28; 34]. Results of research on naturalistic pacing are inconsistent; some show that naturalistic pacing is associated with disability and other poor health indicators [23; 25], while others show the opposite or no association [22; 32; 33]. The cross-sectional nature of existing studies limits us to asking what happens to people *who* pace more or less. To better understand the nature of pacing and guide treatment efforts, studies are needed that examine what happens in terms of symptoms and functioning *when* a person engages in naturalistic pacing. Research that allows for examination of such within-person processes is sparse with one pilot study in osteoarthritis (OA) finding that naturalistic pacing (in this study, defined as going slower and breaking up activities into smaller pieces) was related to more pain, fatigue, and lower physical activity [30].

In the current study, we examined within-person momentary associations between naturalistic pacing and pain and fatigue symptoms in individuals with OA. We hypothesized that increased pain or fatigue would be associated with subsequent increased pacing based on the expectation that naturalistic pacing may be pain or fatigue-contingent (consistent with OPT theory). We also hypothesized that pacing behaviors would have a short-term benefit of subsequent symptom decrease, a pattern consistent with both the OPT model [14], where pacing is a learned behavior reinforced by lower symptom intensity, and by the EC model, where resting is thought to reduce fatigue.

Methods

Design

This is an analysis of data from a multilevel daily process study where participants reported pain and fatigue severity and frequency of use of pacing behaviors five times a day over five days [27]. All study procedures were approved by the Institutional Review Board at the University of Michigan.

Participants

Community-living adults were recruited through public advertisements (newspaper, online, radio, and flyers) in Southeastern Michigan. Details about recruitment have been reported elsewhere [27]. In brief, participants were included if they were age 65 and older, reported at least mild to moderate pain severity overall (a score of ≥ 4 and at least 2 activities with at least moderate pain [17]) on the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain subscale [6] and showed evidence of osteoarthritis in a corresponding knee or hip joint determined by the American College of Rheumatology (ACR) clinical criteria [3; 4]. Participants also needed to meet fatigue criteria by reporting that they felt that they could not get going or that everything they did was an effort [5] for at least 3–4 days in the past week. Participants also needed to have adequate cognitive ability (scoring ≥ 5 on the 6-item screener to identify cognitive impairment) [9], be able to enter ratings on the Actiwatch-Score accelerometer used in the study, and have a consistent, typical sleep schedule (with usual wake-up time before 11am and bedtime before 2am). People were excluded if non-ambulatory (unable to walk with or without an assistive

device), experienced a period of bed-rest for >2 days in the past month, changed medications within the past 2 weeks, had medical conditions that could interfere with symptom ratings or accelerometer data (e.g., rheumatoid arthritis, current cancer treatment, sleep apnea), or if they had other medical reasons for fatigue (abnormal thyroid stimulating hormone or low hemoglobin).

Procedure

Potential participants deemed initially eligible from a phone screening came in for a baseline clinic visit. After written informed consent was obtained, further screening was done to assess eligibility (blood work, ascertainment of clinical criteria for osteoarthritis, and health history) and enrolled participants completed questionnaires. Participants were asked to return for a second clinic visit which included physical performance testing and instruction on how to use the Actiwatch-Score accelerometer with accompanying logbook for use in a 5-day home monitoring period. Participants wore the Actiwatch-Score on their non-dominant wrist for 5 days and were asked to input ratings of pain and fatigue severity and frequency of pacing behaviors into the device 5 times per day as well as record ratings in a logbook. They also reported wake and bed times in the logbook, to assist in actigraphy data processing. A five day sampling period was chosen because it has been deemed an acceptable length of time needed to obtain reliable and valid physical activity data in adult samples [18; 42], without being overly burdensome to participants. Participants were asked to wear the device continuously for the 5 day period except for times when the device could become wet (e.g., showering or swimming). At the end of the home monitoring period, participants were asked to return the device and logbook by mail in a prepaid envelope and were compensated \$80 for all study procedures. There was an overall completion rate of 98% of the symptom reporting. Eighty-six percent of participants had complete symptom reporting (at all 25 time points over the 5 days); the remaining 14% of people had 1–5 responses missing.

Measures

Momentary Measures—Five times per day for 5 days, participants were asked to input symptom and pacing behavior ratings into the Actiwatch-Score accelerometer [Philips Respironics; Mini Mitter, Bend OR]. Rating times occurred at wake-up, 11am, 3pm, 7pm, and bedtime (“lights out”). An audible alarm prompted participants to enter ratings at all time-points except at wake up and bedtimes. Pain and fatigue severity were each rated on a scale of 0 (“no pain/fatigue”) – 10 (“pain/fatigue as bad as you can imagine”) [13; 26]. Fatigue was defined for participants as tiredness or weariness [47]. Pacing behaviors were assessed using three questions based on item stems from the activity pacing subscale of the Chronic Pain Coping Inventory [32] and modified from an earlier study using these questions [30]. Participants were asked to report on the frequency of pacing behaviors in the time since the last reporting period, 4 times per day (excluding wake-up time). On a scale of 0 – 4 (not at all, very little, sometimes, most of the time, always), participants were asked to rate the frequency of use of pacing behaviors in each of 3 questions: 1) *How often have you gone slowly and taken breaks* to do your activities since the last time you rated your symptoms?; 2) How often have you *maintained a reasonable pace* during activities (not too fast or too slow) to reduce the effect of pain on what you were doing since the last time you

rated your symptoms?; and 3) How often did you *break activities into manageable pieces* to do them since the last time you rated your symptoms? Items were summed into a single pacing behaviors scale with a possible range 0 – 12. This scale demonstrated excellent internal consistency (Cronbach's alpha = 0.97) in this sample.

Baseline Demographic and Covariate Measures—The following measures were administered as part of a survey battery at the baseline visit. Demographics of interest included age, sex, race/ethnicity, and marital status. Health status variables of interest included self-reported pain severity in each joint with osteoarthritis, body mass index (BMI); calculated from measured weight (kg)/ height(m)]², illness burden measured as the total number of endorsed symptoms (e.g., headache, stomach pain) out of a list of 41 possible symptoms, and depressive symptoms measured by the short form CES-D [5]. Physical function variables included the Six Minute Walk test [8] and the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) [6] physical disability subscale short form. The Six Minute Walk test is a validated objective physical function measure in which individuals are asked to walk a standard course at their usual pace for six minutes and the distance achieved is recorded. The WOMAC physical function short form scale consists of 7 items and measures perceived difficulty with a variety of activities due to knee or hip pain [46]; it is scored on a scale of 0 – 28; a higher score indicates more physical disability. Pain severity was measured using the WOMAC pain subscale, a five item scale that measures pain severity in different activities due to knee or hip pain. Scores were summed with a higher score indicating more pain [6]. Fatigue severity was measured using the Brief Fatigue Inventory (BFI) severity subscale [26]. This subscale was chosen as it represents a dimension of fatigue which is more highly associated with performance of physical tasks by older adults compared to fatigue interference which is also measured by the BFI [39]. The severity subscale is an average of 3 items from the BFI in which fatigue severity in different contexts is measured on a 0 – 10 scale. Average physical activity over the monitoring period was measured via the Actiwatch-Score accelerometer and was the average daytime activity counts per minute aggregated over the 5 day period.

Data Analysis

Descriptive statistics for all predictor and outcome variables were calculated and examined for distribution normality. Bivariate Pearson correlations were conducted to examine basic between-person associations between key demographic and study variables. Skew and kurtosis values indicated that all variables were sufficiently normally distributed to conduct the primary analyses [44]. To address any modest deviation from normality in the primary analyses, we utilized the “sandwich estimator”, an asymptotically consistent estimator that counteracts problems due to non-normality in the data by generating robust standard errors analyses (as described below) [19; 45].

Multilevel random effects modeling (MLM) was used to test the study hypotheses. This statistical approach was optimal because these data have a hierarchical structure with momentary evaluations of pain, fatigue, and pacing (Level 1) nested within days (Level 2) nested within individuals (Level 3). Using the SAS PROC MIXED procedure, MLM can simultaneously model between- (Level 3) and within-person (Levels 1 and 2) variation and

can account for auto-correlation between adjacent observations. In addition, in MLM, all available data points are used because cases are not eliminated due to missing Level 1 or 2 data. Lastly, MLM allows the modeling of random effects which assumes the independent variable represents a random sample of a larger range of possible values and is generalizable to a broader population compared to a fixed effects analysis. Prior to conducting the MLM analyses, variables were centered based on guidelines for centering data in multilevel statistical procedures [12]. Momentary variables of pain, fatigue, and pacing were person-centered such that values indicate an individual's change in one of these variables from their 5-day average. Between-person variables were sample-centered so that the values indicated an individual's deviation from the sample's mean. All analyses were conducted using SAS software Version 9.3 [38].

To examine how pain and fatigue were associated with subsequent frequency of pacing behaviors, two separate multilevel models were constructed to reduce multicollinearity and because we previously found that pacing is differentially associated with pain and fatigue [29]. In the first model, pacing behavior (the sum of behaviors from the subsequent time point) was entered as the criterion, momentary pain was entered as the main predictor of interest, and average pain on the WOMAC, age, sex, BMI, six minute walk, average activity, illness burden, and depressive symptoms were entered as covariates. We included most of these variables as covariates based on known associations between pacing, symptoms, and disability [22; 25; 31]. Other variables (age, sex, and body mass index) were included based on the fact that they are general demographic variables of interest in studies of pain and activity. The second model was constructed similarly but with fatigue severity as the main predictor variable instead of pain and average fatigue severity on the BFI as a covariate in place of average pain on the WOMAC.

To determine how pacing behavior related to subsequent pain or fatigue severity, two separate multilevel models were constructed. For both models, pacing behaviors was the predictor and the outcomes were either momentary pain or momentary fatigue severity. Both models included all the covariates that were included in the first set of models. Across all models, criterion variables were "lagged" such that the criterion was regressed on predictors from the previous momentary assessment period.

Multilevel models using SAS PROC MIXED do not allow for typical estimations of effects sizes (e.g., R^2 , Cohen's d). We calculated a "pseudo- R^2 " statistic, according to current recommendations [40; 41], for both between-person and within-person effects. This statistic is computed by comparing the variance components of a null or unconditional model (with no predictors) to those of a full or conditional model that contains the predictors of interest to provide an estimate of variance in the criterion accounted for by the model.

Results

Sample Characteristics

Characteristics of the sample ($n = 162$) are shown in Table 1. Results indicated that the sample reported mild levels of pain and stiffness and mild to moderate fatigue. BMI values indicate that the sample was, on average, obese according the United States Centers for

Disease Control standards (e.g., BMI = 30.0). Over half of the sample was married (59%). The sample was mostly Caucasian (83%), followed by African American (11%), Asian (3%), and more than one race (3%). For physical function, the sample walked an average of 1131 feet on the six minute walk test, which is slightly slower than norms from a meta-analysis of studies of community dwelling older adults ($M = 1637$ feet) [7].

Prior to conducting the analyses to test the study hypotheses, we examined the correlations of all the variables to be included in the MLM (Table 2). Momentary pain and fatigue (both averaged across the study period) were highly correlated ($r = .81, p < 0.01$) providing support for the decision to separate pain and fatigue into different models for analysis. The next highest correlations were between illness burden and depressive symptoms ($r = .47, p < 0.01$) and between momentary fatigue and depressive symptoms ($r = .32, p < 0.01$). All other bivariate correlations were of modest magnitude ($r = .30$).

Primary Analyses

How are pain and fatigue associated with subsequent pacing behaviors?—The MLM for pain and fatigue (Table 3) demonstrated similar results in the prediction of subsequent pacing behavior. Both momentary pain and fatigue were significantly and positively associated with reported increases in pacing behaviors in the subsequent time interval. Significant covariates were similar in each model. Higher baseline pain or fatigue, respectively were positively related to pacing activities. Older age (which was marginally significant in fatigue model, $p = .06$), and worse physical function (as indicated by less distance walked during the six minute walk test) were also associated to more pacing behaviors. The model in which pain was examined as a predictor accounted for 11.8% of the between-person and 1.6% of the within-person variance in pacing behavior. The model with fatigue as the predictor accounted for 12.2% of the between-person and 1% of the within-person variance in pacing behavior.

A single model predicting pacing behaviors that simultaneously included pain and fatigue as predictors was constructed post hoc to examine whether considering pain and fatigue together would produce different findings; standard errors, model fit, and significance of individual predictors in the combined model were not substantially different from the separate models. We elected to present the data from the separate models due to previously described conceptual reasons and to mirror the findings of the separate set of models where pacing was the predictor and pain and fatigue the outcomes.

How are pacing behaviors associated with subsequent pain and fatigue?—In the MLM testing the association between pacing behaviors and subsequent pain (Table 4), pacing behaviors were associated with later higher pain and fatigue. Baseline pain severity on the WOMAC and baseline fatigue severity on the BFI were positively related to momentary pain and fatigue, respectively. The model with pacing behavior as the predictor of pain accounted for 24.8% of the between-person variance and 10.6% of the within-person variance in pain. The model with pacing behavior as the predictor of fatigue accounted for 23.9% of the between-person and 9.3% of the within-person variance in fatigue.

Supplementary Analyses

How are self-reported pacing behaviors associated with physical activity level?—The Actiwatch-Score is an accelerometer that collects objective physical activity data in the form of activity counts. We conducted analyses to examine how self-reported pacing behaviors were related to concurrent physical activity. In two multilevel models (controlling for covariates of baseline pain, baseline fatigue and all other variables in models in Table 3), when self-reported pacing behaviors increased, concurrent physical activity (average activity counts/minute) decreased [β (pacing) = -4.18 ; $p = .01$] and the percentage of time spent immobile increased [β (pacing) = $.52$; $p = .01$]. For every one unit increase in pacing, there was an approximately 4 point decline in activity counts per minute and .5 percent decrease in time spent immobile. This suggests that when people report increased pacing behaviors, their physically active level dropped.

Discussion

We sought to further the understanding of activity pacing by examining how spontaneous, untrained pacing in daily life, or *naturalistic pacing*, is associated with pain and fatigue symptoms within days in individuals with OA. Activity pacing is often taught as a behavioral strategy with underlying principles from OPT or EC models; therefore, we related our results to these models. Our findings support the distinction of naturalistic pacing from programmatic (taught) pacing in two ways based on OPT and EC models—1) symptom-contingency and 2) reinforcement or “pay-off” of the behavior.

Symptom Contingency

Our findings support the contention in OPT that naturalistic pacing is symptom-contingent. That is, individuals may be reacting to increased pain or increased fatigue by pacing. Although not large effects, older age and lower physical function were significant covariates of the association between symptoms and subsequent pacing behaviors. The positive association between symptoms and subsequent pacing behavior remained above and beyond the effects of these variables. Because of this symptom-contingency, these findings also suggest that naturalistic pacing may be maladaptive, which are consistent with other studies that show pacing is associated with disability [23; 25]. Specifically, disability may be promoted by this symptom-contingent pattern of reducing activity in response to pain or fatigue which can lead to inactivity, physical deconditioning, and reduced physical capacities over time [14]. When teaching pacing, a key principle based on OPT is to disassociate symptoms from activity so that behaviors are not symptom contingent but rather task- or time-contingent [35]. For instance, using ‘time-based’ activity pacing, in which activity and rest breaks are practiced on a time schedule, within-day pacing behaviors would be consistently practiced across the day; behaviors would not fluctuate based on pain or fatigue. Thus, while naturalistic pacing appears to be symptom-contingent, programmatic pacing (if practiced as instructed), would not be symptom-contingent.

Reinforcement of Pacing

We found that naturalistic pacing was associated with later increases in pain and fatigue. This seems counterintuitive as symptom reduction might be a plausible reinforcing and

immediate “pay-off” to pacing (e.g. resting or going slow might result in short-term pain decreases); this concept is consistent with both OPT and EC models. However, this study considered only symptom reduction, a type of negative reinforcement, but did not assess myriad types of positive reinforcement, such as attention from others [15]. We also did not measure between-person factors that might have revealed individual differences in reinforcement of pacing. In addition, naturalistic pacing may reflect a larger, more complex interplay of factors not completely captured in our models. For example, pain, fatigue, and pacing behaviors all increase over the day; these concomitant increases may be influenced by other factors such as comorbid health issues, medication effects, social context, momentary mood, or habitual daily routines [43]. Further research is needed to more comprehensively measure factors that potentially influence how pacing is used in everyday life.

The findings suggest that the size of moment-to-moment associations between symptoms and pacing are quite small; however, effect sizes can be difficult to interpret in momentary process studies such as this and must be considered in the context of their potential real-world impact. The importance of an effect is not wholly dependent on the size of the effects and the meaning of small effects has been discussed extensively [1; 10; 36; 37]. Careful consideration of the importance of small effects may be particularly true in cases where small effect events occur many times [1; 36; 37]; small effects may accumulate over many occurrences to show consequential effects over time. For example, although the momentary association between pain and subsequent pacing is small, over hours, days, and years, that small association may have larger consequences in terms of coping strategy selection, emotional distress, and physical functioning.

Interestingly, our models predicting pacing behavior explained a small amount of the variance in pacing, suggesting that other unmeasured factors, such as motivational factors, may be major contributors to pacing behaviors. This study, unfortunately, did not assess motivations for pacing, which could differ between people and within a person. One way to conceptualize motivation for pacing behaviors is in terms of two motivational systems - the behavior inhibition (or avoidance) system (BIS) and the behavioral activation (or approach) system (BAS). The BIS, enacted through withdrawal behaviors, exists primarily for self-protection [9]; whereas the BAS is related to approach behaviors and seeking reward and pleasure [9]. Consistent with BIS, these findings could be interpreted in the context of the fear-avoidance model, whereby catastrophic interpretations of pain sensations create fear of pain, which can lead to a cycle of habitual activity avoidance, disuse, and disability [43]. In this sample, lower levels of physical function (six-minute walk) were related to high pacing. Although we cannot infer causal direction, these findings might indicate a process where lower levels of activity for those who fear pain, compounded over years, contributed to poorer physical functioning.

In contrast to pacing activity for self-protection and pain avoidance, people may pace activity in pursuit of a goal, reflecting a BAS motivational framework. This notion is reflected in many different descriptions of activity patterns in chronic pain, including task/activity persistence [20; 21], acceptance and commitment therapy [11], chronic pain acceptance [25], and committed action [24]. The BIS/BAS framework might be helpful for

conceptualizing different motivations for pacing because existing activity pacing interventions can be thought of as working to shift the dominant motivational framework of the patient from BIS- to BAS-based.

Limitations and Future Directions

Our findings can be generalized only to older adults with osteoarthritis; the chronic condition sampled is likely an important distinction as pacing behaviors may vary by condition [28]. We did not screen participants to determine whether they had ever participated in a pacing program; however, extensive experience with this patient population indicates a very low likelihood. Consequently, we expect rates of programmatic pacing to be minimal in this study sample. We currently know very little about the temporal aspects of pacing, such as how long it might take for pain to impact pacing and vice versa. Future research should explore the optimal time frame for assessing the temporal associations between activity and symptoms to more fully understand the momentary processes of pacing. The fact that self-reported pacing was found to be related to lower levels of concurrent physical activity does not provide any information on whether pacing is related to better overall productivity or task persistence. This might suggest that our measure of pacing captured behavioral aspects of resting and going slowly more than breaking up tasks or keeping up a steady pace. Indeed the fact that our measure of momentary pacing combined a number of distinct facets of pacing, each of which may have different effects on symptoms and functioning, could be considered a limitation of the study. Further, pacing (and the validity of OPT vs. EC models) may play a different role in osteoarthritis symptoms compared to other conditions like fibromyalgia or multiple sclerosis, given their different symptom burdens. Future research should examine associations between pacing behaviors and other key variables in samples of individuals with different chronic conditions. Only one out of the three items on our assessment of naturalistic pacing specified a goal of pacing behavior, which was to reduce the effect of pain. Therefore, it remains unclear whether naturalistic pacing was done to achieve a goal. Because the specific goal of the pacing behavior (i.e., pain reduction, increased energy, increased productivity, attainment of a valued activity goal) has been identified as a key contextual variable that may influence the importance and effects of pacing, future research should include items that assess the goal(s) of each pacing behavior. The focus of the OPT treatment of pacing behavior is to encourage patients to switch from a primary goal of symptom management (pain and fatigue reduction) to activity management and valued goal achievement (e.g., return to work, increased social participation). Yet we measured pain and fatigue as the primary dependent variables in this study. Future research should also consider measures of activity and participation as outcomes.

Some have questioned the utility of teaching pacing as an adaptive strategy based on conclusions that pacing may be maladaptive and contribute to increased disability [22]. However, it may not be possible to draw conclusions about the utility of programmatic pacing based on findings from studies of naturalistic pacing. That is also true in the case of this study. Although our ultimate aim is to inform the efforts of pacing-based interventions, our results only pertain to potential areas for improvement in what we have observed about naturalistic pacing.

Conclusion

In conclusion, our study showed strong associations between naturalistic pacing and symptoms as experienced over time. Naturalistic pacing appears to be symptom-contingent and does not appear to be reinforced by symptom reduction (as symptoms increased with increased use of pacing). Future research is needed to better understand naturalistic pacing in OA in different chronic conditions with different symptom profiles, which would provide important information about behavioral patterns that may be targets for condition-specific intervention.

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Table 1

Demographics of Participants with Symptomatic Knee or Hip Osteoarthritis

Variable	N	Mean	SD	Range
Age	162	72.02	5.89	65–90
Women, %	162	61.7		
Marital Status	161			
Single, never married (%)	5	3.1		
Married	95	58.6		
Divorced	33	20.4		
Widowed	28	17.3		
Caucasian, %	134	82.7		
Body Mass Index	160	30.31	5.68	20–52
Illness Burden	160	9.29	4.28	0–24
CES-D Depressive Symptoms	162	11.25	7.95	0–35
WOMAC Pain	161	8.59	3.11	2–20
WOMAC Stiffness	160	3.63	1.54	0–8
WOMAC Physical Disability-Short Form	159	10.79	4.12	0–22
Brief Fatigue Inventory Total	161	4.54	2.02	.25–9
Six Minute Walk (feet)	161	1131.15	251.97	265–1770
Average Activity (activity counts per minute)	159	324.48	87.01	549.17
Momentary Pain*	162	3.22	1.70	0–8.9
Momentary Fatigue*	162	4.0	1.78	0–8.5
Momentary Pacing Behaviors*	162	5.81	2.53	0–12

Note.

CES-D = Center for Epidemiological Studies Depression Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; Illness Burden = # self-report health problems/symptoms, 41 possible;

* average of all momentary self-report ratings over the study period

Table 2

Bivariate Pearson Correlations of all variables in MLMs

	Momentary Fatigue	Momentary Pacing	WOMAC Pain	BFI Fatigue	Age	BMI	Six Minute Walk	Average Activity	Illness Burden	CES-D
Momentary Pain [†]	.81**	.27*	.49**	.47**	-.04	.15	-.15	.03	.29*	.29*
Momentary Fatigue [†]	1	.28*	.34*	.53**	-.06	.09	-.16	.01	.31**	.32**
Momentary Pacing [†]		1	.20	.22*	.18	.07	-.27*	-.08	.15	.18
WOMAC Pain			1	.49**	-.15	.17	-.21	.09	.33**	.23*
BFI Fatigue				1	-.07	.05	-.14	-.03	.49**	.003
Age					1	-.25	-.22	-.21	-.004	.007
BMI						1	-.20	-.11	-.01	.003
Six Minute Walk							1	.25*	-.08	-.05
Average Activity [†]								1	-.001	-.05
Illness Burden									1	.47**

Note.

CES-D = Center for Epidemiological Studies Depression Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; BFI = Brief Fatigue Inventory; BMI = body mass index; Illness Burden = # self-report health problems/symptoms, 41 possible;

[†] Average value over the week long home monitoring period;

* $p < 0.05$,

** $p < 0.01$

Table 3

Multilevel Regressions of Momentary Associations Between Changes in Pain and Fatigue and Subsequent (Lagged) Changes in Activity Pacing

Covariance Parameter	Subject	Random Effects							
		Pain→Pacing			Fatigue→Pacing				
		Estimate	SE	Z	P	Estimate	SE	Z	P
Intercept CS	ID	5.25	.66	7.94	<.0001	5.26	.66	7.97	<.0001
CS	ID	-0.02	.0006	-32.29	<.0001	-.02	.0005	-32.47	<.0001
CS	DAY(ID)	1.00	.12	8.68	<.0001	.99	.11	8.75	<.0001
Residual	•	3.41	.11	32.29	<.0001	3.40	.11	32.47	<.0001

Effect	Fixed Effects							
	Pain→Pacing			Fatigue→Pacing				
	β	SE	t	P	β	SE	t	P
Intercept	5.70	.32	17.74	<.0001	5.81	.31	18.48	<.0001
Level 1 (df = pain 2699, fatigue 2720)								
Momentary pain/fatigue	.11	.03	4.16	<.0001	.11	.03	3.74	.0002
Level 3 (df = pain 145, fatigue 146)								
WOMAC pain severity	.08	.07	1.26	.21				
BFI fatigue severity					.16	.12	1.31	.19
Age	.07	.04	1.95	.05	.07	.04	1.93	.06
Sex	-.06	.41	-.13	.89	-.17	.40	-.43	.67
Body Mass Index	.02	.04	.57	.57	.03	.04	.71	.48
Six Minute Walk (feet)	-.002	.001	-2.31	.02	-.002	.001	-2.37	.02
Average Activity	.001	.002	.51	.61	.001	.002	.47	.64
Illness Burden	.02	.05	.53	.60	.02	.05	.47	.64
CES-D	.04	.03	1.60	.11	.03	.03	1.08	.28

Note. A compound symmetry matrix was used to model the error variance on the DV. Level 1 variables are person-centered. Level 3 variables are sample-centered.

ID = subject identifier; CES-D = Center for Epidemiological Studies Depression Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; Illness Burden = # self-report health problems/symptoms, 41 possible);

* average of all momentary self-report ratings over the study period

Table 4
 Multilevel Regressions of Momentary Associations Between Changes in Activity Pacing and Subsequent (Lagged) Changes in Pain and Fatigue

Covariance Parameter	Subject	Random Effects							
		Pacing→Pain		Pacing→Fatigue					
		Estimate	SE	Z	p	Estimate	SE	Z	p
Intercept CS	ID	2.07	.27	7.69	<.0001	2.22	.28	8.03	<.0001
CS	ID	.08	.0003	32.74	<.0001	-0.03	.001	-32.83	<.0001
CS	DAY(ID)	.33	.04	7.90	<.0001	.21	.05	4.13	<.0001
Residual	•	1.41	.04	32.74	<.0001	2.31	.07	32.83	<.0001

Effect	Fixed Effects							
	Pacing→Pain		Pacing→Fatigue					
	β	SE	t	p	β	SE	t	p
Intercept	3.25	.20	16.67	<.0001	3.92	.21	18.41	<.0001
Level 1 (df = 2759, 2778)								
Activity Pacing	.10	.02	4.78	<.0001	.11	.03	4.61	<.0001
Level 3 (df = pain 145, fatigue 146)								
WOMAC pain severity	.22	.06	3.75	.0003				
BFI fatigue severity					.41	.07	5.82	<.0001
Age	.005	.02	.21	.83	-.004	.02	-.17	.87
Sex	-.02	.26	-.07	.95	.32	.26	1.20	.23
Body Mass Index	.02	.03	.78	.44	.01	.03	.59	.56
Six Minute Walk (feet)	-.0005	0.001	-.90	.37	-.001	.001	-1.05	.30
Average Activity	.002	.001	1.27	.21	.001	.001	1.13	.26
Illness Burden	.03	.03	.91	.36	.02	.04	.47	.64
CES-D	.03	.02	1.84	.07	.01	.02	.59	.56

Note. A compound symmetry matrix was used to model the error variance on the DV. Level 1 variables are person-centered. Level 3 variables are sample-centered.

ID = subject identifier. CES-D = Center for Epidemiological Studies Depression Scale; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; Illness Burden = # self-report health problems/symptoms, 41 possible);

* average of all momentary self-report ratings over the study period

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