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Static and Dynamic Pain Sensitivity in Adults with Persistent Low Back Pain: Comparison to Healthy Controls and Associations with Movement-Evoked Pain Versus Traditional Clinical Pain Measures

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Abstract

Objectives—Despite its impact, individual factors associated with persistent low back pain (LBP) remain poorly understood. This study investigated static and dynamic pain sensitivity in adults with persistent LBP vs healthy controls; and investigated associations between pain sensitivity and three clinical pain measures: recalled, resting, and movement-evoked pain (MEP).

Materials and Methods—A lifespan sample of 60 adults with persistent LBP and 30 age-/sex-matched controls completed four laboratory sessions. Static pain sensitivity (pressure pain (PPT), heat pain threshold) and dynamic pain sensitivity (heat pain aftersensations (AS), temporal summation (TS) of second heat pain) were measured. Demographic and clinical factors collected were education, global cognition and perceived health. Resting and recalled pain were measured via questionnaire, and MEP via the Back Performance Scale.

Results—LBP participants demonstrated lower PPT remotely (hand; $F_{1,84} = 5.34$, p=.024) and locally (low back; $F_{1,84} = 9.55$, p =.003) and also had higher AS ($F_{1,84} = 6.01$, p=.016). Neither static nor dynamic pain sensitivity were associated with recalled pain (p>.05). However, static pain sensitivity (local PPT) explained an additional 9% variance in resting pain, while dynamic pain sensitivity (AS, TS) explained an additional 10–12% variance in MEP.

Discussion—This study characterized pain sensitivity measures among individuals with persistent LBP and suggests static pain sensitivity plays a larger role in resting pain while dynamic

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pain sensitivity plays a larger role in MEP. Future studies will confirm these relationships and elucidate the exent to which changes in static or dynamic pain sensitivity predict or mediate clinical pain among adults with persistent LBP.

Keywords

low back pain; pain sensitivity; movement-evoked pain

INTRODUCTION

Low back pain (LBP) accounts for more years lived with disability than either cardiopulmonary or cognitive disease, making it the most disabling health condition in the world.[1–4] Patients with LBP also incur health-related costs twice that of patients without LBP.[2,4] Yet despite high health-related costs, low back pain interventions are only modestly effective. The majority of patients with LBP who receive clinical care will report persistent pain and disability one year later.[5,6] Modest effects of LBP interventions can be traced back to our knowledge gap in individual factors that increase risks for persistent pain and disability among patients with LBP.[7]

A promising individual factor that may predict persistent pain and disability is pain sensitivity – i.e., the function of peripheral, spinal, and supraspinal pain pathways. Pain sensitivity exists in two forms: 1) static, or pain experienced at a single time point (e.g., pain threshold); and 2) dynamic, or changes in pain over time in response to sustained or repeated stimuli (e.g., pain facilitation via ramp and hold, temporal summation of second pain). Pain sensitivity is assessed using quantitative sensory testing (QST) paradigms which employ experimentally-evoked pain stimuli (e.g., pressure or heat) and follow the participant's response. Such paradigms were born out of decades of animal literature and are well established;[8–10] and evidence suggests that pain sensitivity via QST is not only altered among individuals with persistent pain, but also predictive of their suboptimal recovery outcomes.[11–13] Moreover, many QST paradigms are readily translatable to clinical care.[14,15] However, for individuals with LBP the presence and implications of altered pain sensitivity are less clear. A narrative review found pain sensitivity among individuals with persistent LBP was either similar to healthy controls, or dependent upon region or test paradigm.[16] A meta-analysis found only weak associations between pain sensitivity and clinical pain ratings (resting pain intensity) in people with LBP.[17] In contrast, a recent meta-analysis of prospective studies and RCTs found pain sensitivity across many pain conditions, including LBP, was predictive of clinical pain ratings.[11]

Equivocal findings for pain sensitivity and clinical pain ratings for persistent LBP may be explained by two factors. First, pain sensitivity during persistent LBP is complex and multifactorial, and influenced by a number of test conditions and individual factors. Second, associations between pain sensitivity and clinical pain ratings may depend upon how clinical pain is defined. The two predominant clinical pain measures are recalled pain and resting pain, which measure pain by administering a questionnaire while the participant or patient is in a rested state (i.e. not moving). However, recent research suggests that a third measure may be more appropriate than resting pain for research and clinical care[18] – movement-

evoked pain (MEP). MEP is produced by movement in real-time, and can be measured either through isolated movements like knee flexion[19] or gross functional movements like walking; [20] which may mimic the pain experience of many people with chronic pain (i.e., it hurts when I move). Unfortunately, pain sensitivity studies which previously investigated associations with clinical pain almost exclusively measured recalled and resting pain, rather than MEP.

Therefore, our study purpose was to elucidate relationships among static and dynamic pain sensitivity, and recalled, resting, and MEP ratings for individuals with persistent LBP. Our first aim was to compare pain sensitivity assessed via QST among individuals with persistent LBP to age- and sex-matched individuals without pain. Our second aim was to compare associations between pain sensitivity and clinical pain measures; specifically, common clinical pain measures like recalled pain and resting pain via questionnaire, versus MEP assessed via physical performance test. The overarching goal of this line of research is to elucidate the extent to which altered pain sensitivity mediates clinical pain ratings and recovery among patients with persistent LBP.

MATERIALS & METHODS

Study Procedures

In addition to matched control individuals, this analysis includes data from adults with persistent low back taken from a single arm, mechanistic trial comparing age-specific effects of high amplitude transcutaneous electrical nerve stimulation (TENS) on pain sensitivity and clinical pain measures.[21] Importantly, we found no differences in pain sensitivity or clinical pain response by age group, when older adults received TENS parameters appropriately dosed.[21] Also, TENS effects were found to be episodic versus cumulative, meaning pain sensitivity and clinical pain response were within session but not across sessions.[21] Therefore, we included all sessions where both static and dynamic pain sensitivity were measured (n=4), but only used pre-TENS intervention data (FIG 1). Participants completed up to 2 sessions per week, which is similar to clinical implementation of TENS as well as a seminal TENS efficacy trial performed among individuals with CLBP. [22] Importantly, study procedures were the same for adults with persistent LBP and matched control individuals.

At baseline, participants provided demographic information, cognitive tests, and reaction time tests; as well as outcomes data by questionnaire for resting and recalled pain. Participants then performed a physical performance test to capture MEP. Finally, participants completed a battery of static and dynamic pain sensitivity tests including heat and pressure pain threshold, heat pain aftersensations, and temporal summation of second heat pain (TS). At three subsequent sessions, participants completed only the static and dynamic pain sensitivity tests.

Participants

To ensure a representative sample of 60 individuals with persistent LBP across the lifespan, we employed a purposive sampling method to stratify to the following age group quotas: Young (18–39 years old), middle-aged (40–56 years old) and older (57–79 years old). These

age group ranges were determined *a priori* based on previous research.[23,24] Upon reaching 20 participants for a particular age group, that group was closed to further enrollment. Age group quotas allowed us to recruit an equal number of participants across the lifespan, which aligns with current NIH initiatives.[25]

Thirty pain-free, age-matched (+/− 2 years) individuals were also included in this study and were sex-matched to participants with persistent LBP. Sex matching was based on a binary question of whether the participant identified as male or female. All participants were enrolled at the University of Florida Health Science Center between September 2013 and October 2014.

Eligibility Criteria—For individuals with persistent LBP, inclusion criteria included a primary complaint of LBP for at least three months prior to study enrollment and an average daily LBP pain intensity equal to or greater than 40/100 at worst on a zero to 100 scale (0='no pain'; 100='worst pain imaginable'). The inclusion criterion for pain-free individuals was no LBP or other persistent pain condition in the three months preceding study enrollment.

Exclusion criteria for both individuals with persistent LBP or pain-free were as follows: 1) symptoms of lower extremity nerve root involvement such as motor weakness and sensory disturbance; 2) prior surgery for low back musculoskeletal pain; 3) current use of opioids; 4) comorbidities including uncontrolled hypertension, diabetic neuropathy, circulatory disorders interfering with activities of daily living, cardiac event history (e.g. myocardial infarction), or epilepsy; 5) implanted cardiac pacemaker; 6) psychiatric-related hospital admission within the past year; 7) pregnancy; or 8) cognitive impairment based on a score lower than 23 on the Mini Mental State Examination (MMSE).[26] Specific to individuals with persistent LBP, exclusion criteria also included pain not resulting from trauma - like a car accident, work accident, or fall; and no pain treatment by a health care professional within the past month.

Study Measures

Demographic Factors—Participants provided baseline information on a demographic questionnaire that included age (continuous), identified sex (female/male), education level (high school versus college attendance), and perceived health (poor to fair versus good to excellent).

Cognitive Factors—Next, global cognition was tested using the Mini Mental State Examination (MMSE).[26] As mentioned, participants with MMSE scores lower than 23 were excluded from the study to prevent the enrollment of those with cognitive impairment. However, MMSE scores were also included as a covariate in analyses of pain sensitivity secondary to the potential to influence quantitative sensory testing performance.

Pain Sensitivity Factors—Static and dynamic pain sensitivity test locations are illustrated in Figure 2.

Static Pain Sensitivity Factors: Pressure pain threshold (PPT) was assessed by applying 1 kilogram-force per centimeter-squared per second to three anatomical regions using a Wagner Force Ten FDX 25 Digital Force Gauge™ (Wagner Instruments, Greenwich, CT). PPT was first assessed remotely using the thenar eminence of the right hand. In previous studies, participants with pain conditions demonstrated decreased PPT at remote locations compared to healthy participants, which was considered an indication of generalized changes in pain sensitivity.[21,39,40] PPT was then assessed locally in the region of axial CLBP at bilateral posterior superior iliac spines (PSIS). Participants indicated the first onset of pain for each trial, and a total of three trials were performed at each anatomical site. Remote and local PPT trials were averaged together to arrive at a single remote and local PPT value.

Heat pain threshold (HPT) was assessed at the volar aspect of right forearm in the C8 dermatomal distribution using a $3cm^2$ thermode connected to a PATHWAY Model Advanced Thermal Stimulator (ATS) (Medoc Advanced Medical Systems, Ramat Yishai, Israel). Initial temperature was 33°C and increased at a rate of 1°C/sec, with a maximum allowable temperature of 50°C. Participants indicated when they first perceived the sensation of heat pain, and the temperature (degrees Celsius (°C)) corresponding to that pain was then recorded.

Dynamic Pain Sensitivity Factors: Two dynamic pain sensitivity factors were assessed, heat pain aftersensations and temporal summation of second heat pain (TS). Both paradigms were assessed using a $3cm^2$ thermode connected to a PATHWAY Model Contact Heat-Evoked Potential Stimulator (CHEPS; Medoc Advanced Medical Systems, Ramat Yishai, Israel.

To measure heat pain aftersensations, participants first completed stimulus/response testing of 15 second stimuli at three temperatures (46°C, 47°C, 48°C) at the plantar aspect of the left foot to determine the temperature closest to a pain intensity level of 50/100 on a zero to 100 numeric pain rating scale (NPRS), where zero is ‘no pain’ and 100 is ‘worst pain imaginable. The corresponding heat stimulus was then applied for 30 seconds (10°C/sec on-ramp time) to the plantar aspect of the right foot posterior to the first metatarsophalangeal joint. Participants rated their pain intensity every five seconds using the same NPRS. Finally, heat pain aftersensations were calculated immediately following the 30-second application of heat stimulus. As the temperature was decreased at a rate of 10°C/second to neutral temperature (33°C), participants continued to rate their pain intensity every two seconds for a total of 10 seconds using the NPRS, which provided an indication of aftersensations after removal of the painful stimulus. The resultant pain ratings (n=5) were then used to calculate a trapezoidal area under the curve (AUC), which quantified the magnitude of heat pain aftersensations for each individual.[27]

For TS, five consecutive heat pulses delivered for 1 second with a 3 second interval (0.33Hz stimulus frequency) were delivered at 48°C to the plantar aspect of the right foot just anterior to heat pain aftersensations. Participants were instructed to rate the second pain experienced for each pulse after the initial onset of heat. Ratings of the first heat pulse were subtracted from ratings of the fifth heat pulse to calculate a TS score.[28]

Clinical Pain Measures—Recalled and resting pain were measured using the Brief Pain Inventory Short Form (BPI). Traditionally, the BPI is used as a measure of 'daily pain intensity' which is the mean of four ratings: best and worst pain intensity over the previous 24 hours, average pain intensity, and present pain intensity. However for the purpose of this analysis, 'recalled pain' was considered the mean of best and worst pain intensity over the previous 24 hours plus average pain intensity. Similarly, 'resting pain' was deemed be the rating for present pain intensity, since ratings were taken while the patient was in a resting position for almost 30 minutes. Each rating is scored using an 11-point numeric pain rating scale, with zero meaning "no pain" and 10 meaning "worst pain imaginable." The BPI has been deemed a valid and reliable measure of musculoskeletal pain.[29]

MEP intensity was assessed through functional tasks of the Back Performance Scale (BPS).[30] The BPS determines how CLBP affects physical performance and consists of five physical tests requiring spinal movement to complete a particular task. The five physical tests are simulation of putting on socks, picking up a piece of paper, bending to touch toes knee straight, transitioning from supine lying to long-sitting without hand assistance, and repetitive 5 kilogram box lifting from floor to waist for one minute. Participants rated their 'pain rating at worst' during each task on a zero to 100 NPRS. A MEP intensity composite score was created for each individual by averaging pain ratings for the five tasks, with higher scores indicating greater MEP intensity. Use of a MEP composite score was determined *a priori* and deemed appropriate since 1) inter-correlation of pain across the five tasks was between 0.60 and 0.85; 2) using an average of pain ratings rather than a single pain rating reduces the error variance; and 3) using a composite score increases capacity for explained variance to examine associations.[31]

Statistical Analysis

Analyses were completed using IBM® SPSS® Statistics software, Version 25 (2017, IBM® Corp; Armonk, NY). A cumulative rating was determined for each static and dynamic pain sensitivity test by averaging ratings across all four individual test sessions (FIG 1). Group differences for demographic continuous factors (e.g., age) were assessed using independent student t testing. Because of sample size differences, Mann-Whitney U nonparametic testing was used to confirm group differences in the presence of variance inequality (Levene's test). Chi-square test assessed group differences in categorical factors (e.g. education). Alpha level was set at p=.10 in determination demographic factor covariates, and p=.05 for final analyses.

Aim 1: Group Differences in Static and Dynamic Sensitivity—Independent student t testing and Mann-Whitney U nonparametic testing were used to assess unadjusted group differences in static and dynamic pain sensitivity measures. Next, analysis of covariance (ANCOVA) was used to calculate adjusted group mean differences by covarying demographic factors that differed between groups (p<.10). Prior to analysis, factors were z-transformed to allow comparisons across pain sensitivity factors.

Aim 2: Associations between Static and Dynamic Pain Sensitivity and Clinical Pain Measures—Among participants with persistent LBP only, bivariate associations

between demographic factors, pain sensitivity factors, and clinical pain measures were assessed using nonparametric Spearman's rank correlation tests. Separate multivariate hierarchical regression models were then created via ordinary least squares regression modeling to examine pain sensitivity associations with clinical pain after adjusting for demographic factors (**Aim 2**). Demographic factors associated with either pain sensitivity or clinical pain measures in the previous analysis ($p<.10$) were entered into the first block, and the corresponding pain sensitivity factor into the second block. This allowed for examination of unique variance (R^2 change) of pain sensitivity factors after controlling for demographic factors. Standardized regression coefficients (beta) were used to compare strength of association for factors in the final block. We employed *a priori* cutoff rules for intercorrelation ($r<.70$), tolerance ($r>.20$), and variance inflation (<4) in order to confirm model stability and absence of multicollinearity.

RESULTS

Sixty participants with persistent LBP and 30 age- and sex-matched pain-free control participants were enrolled. Control participants were validated based on near zero ratings on clinical pain measures. In addition, matching was validated based on similar age and sex ($p>.05$). Demographic factors that differed based on *a priori* alpha level cutoff ($p=.10$) were education ($p=.036$), global cognition ($p=.06$), and perceived health ($p=.002$) (Table 1). On average, participants completed the four study sessions in 13.7 days, with an average of 4.6 days between sessions.

Aim 1: Group Differences in Static and Dynamic Sensitivity

With exception of TS, unadjusted means for static and dynamic pain sensitivity differed between groups ($p<.05$; FIG 3a). After adjusting for education, global cognition, and perceived health (FIG 3b), group differences no longer existed for heat pain threshold ($F_{1,84} = 1.61$, $p=.208$); but remained for remote PPT (thenar; $F_{1,84} = 5.34$, $p=.024$), local PPT (PSIS; $F_{1,84} = 9.55$, $p =.003$) and heat pain aftersensations ($F_{1,84} = 6.01$, $p=.016$).

Aim 2: Associations between Static and Dynamic Pain Sensitivity and Clinical Pain Measures

Bivariate Associations (Table 2)—Among the persistent LBP group only, associations with demographic factors and pain sensitivity factors were variable (Table 2a). Age and sex were not associated with any clinical pain measure ($p>.10$); while education, global cognition, and perceived health were negatively associated with all three clinical pain measures (Spearman $\rho=-.274$ to $\rho=-.445$). Only TS was associated with recalled pain (Spearman $\rho=-.351$), while both local PPT and TS were associated with resting pain (Spearman $\rho=-.291$ to $\rho=-.436$) (Table 2b). With the exception of remote PPT, all static and dynamic pain sensitivity measures were associated with MEP (Spearman $\rho=-.307$ to $\rho=-.488$).

Multivariate Associations (Table 3)—Perceived health was the lone demographic factor associated with all clinical pain measures and was often the strongest factor in multivariate models. After adjusting for demographic factors, TS was no longer associated

with recalled or resting pain (p>.05). However, local PPT remained associated with resting pain, explaining an additional 9% variance. Static pain sensitivity factors (local PPT and heat pain threshold) were no longer associated with MEP (p>.05), but both dynamic pain sensitivity factors (heat pain aftersensations and TS) remained associated; explaining an additional 10% and 12% variance in MEP, respectively.

DISCUSSION

This study characterizes the alterations in static and dynamic pain sensitivity among adults with persistent LBP and suggests novel information about associations between pain sensitivity and clinical pain measures. The majority of pain sensitivity measures were altered among adults with persistent LBP versus age- and sex-matched controls, even after adjusting for demographic factors. Compared to the scarcity of factors associated with a traditional measure like recalled pain, bivariate associations existed between pain sensitivity and MEP in all but one instance. Perhaps the most important findings relate to association specificity: a static pain sensitivity measure was associated with a static clinical pain measure (resting pain), while dynamic pain sensitivity measures were associated with a dynamic clinical pain measure (MEP). Previous work by our group and others suggest pain sensitivity plays an important role in the Fear-Avoidance Model of pain persistence and functional consequences (e.g., activity-avoidance) – above and beyond psychological distress factors alone.[32–34] Current findings add to this work by suggesting dynamic pain sensitivity is associated with MEP, which is a logical activity-avoidance precursor. Future work will validate this assertion and investigate the broader role of dynamic pain sensitivity and MEP in the Fear-Avoidance Model pathway, including modifiability to facilitate persistent pain recovery.

Earlier reviews have reported inconsistent findings across studies examining pain sensitivity differences in people with LBP versus healthy controls, and/or in associations between pain sensitivity and clinical pain measures.[16,17] Methodological features of our study may help explain our results in the context of previous findings. First, since we were interested in the extent to which static versus dynamic pain sensitivity were associated with clinical pain measures, we used relatively homogenous QST parameters compared to earlier work.[17] Second, earlier reviews found the majority of study sample sizes were low, and noted lack of power as a potential explanation for conflicting findings.[16,17] Third, not all previous studies employed age- and sex- matching, or sampled across the lifespan as federal initiatives for clinical research now mandate.[25] A cohort commonly neglected in LBP studies overall are older adults, which previous work from our group has shown to be susceptible to senescence-related changes in the pain sensitivity system.[24,35–37] Finally, there are multiple differences between previous studies and this study in QST measures used to assess pain sensitivity, most notably our dynamic pain sensitivity measures. TS is commonly tested in the upper extremity using varying modalities (e.g., mechanical, electrical); whereas we assess TS in response to heat pain at the plantar aspect of the foot. Similarly, we tested heat pain aftersensations which are not common to pain sensitivity studies in LBP. Previous work found associations with widespread clinical pain and argue strongly for using pain aftersensations to measure centrally-mediated pain facilitation.[38–40]

Perhaps the biggest difference between this study and previous work in LBP was the inclusion of MEP. MEP is a hallmark of persistent musculoskeletal pain conditions and has higher severity than resting pain.[41] Also, previous studies found musculoskeletal pain interventions to be more effective for reducing MEP than resting pain.[42,43] However, only a few studies have tested pain sensitivity and used those measures to predict MEP; and of these studies, the majority occurred in regions outside the low back. Our group induced delayed onset muscle soreness (DOMS) to the shoulder of healthy adults, and found that a proxy of dynamic pain sensitivity (suprathreshold heat pain) predicted MEP with endrange shoulder abduction.[44] Similarly, a knee arthroplasty study by Rakel et al.[19] found pre-operative static pain sensitivity (punctate pain sensitivity) predicted postoperative MEP with endrange knee flexion. In both studies, pain sensitivity measures predicted MEP, but not resting pain.[19,44]

Importantly, these preceding studies isolated movement to single plane joint motion like shoulder abduction and knee flexion. Perhaps more informative are studies investigating MEP in the context of physical performance; which has been consistently linked to disability and downstream health outcomes.[45,46] Wideman et Al.[20] found older adults with knee osteoarthritis experienced over a 100% increase in their pain with the six-minute walk test (6MWT), and pain with the 6MWT was uniquely predicted by temporal summation of punctate pain. More recently, a cross-sectional study of widespread chronic pain by Woznowski-Vu et Al.[47] found pain with a lifting task was uniquely predicted by temporal summation of punctate pain, but not static pain sensitivity (PPT). Notably, the Back Performance Scale used in this study includes a lifting task. Combined findings from the current study and previous studies reveal two themes related to pain sensitivity and MEP: 1) pain sensitivity is more commonly associated with MEP than resting pain; [19,44] and 2) MEP is more commonly associated with dynamic pain measures versus static pain measures. [20,44,47] Thus, a priority of future research is to include both static and dynamic pain sensitivity measures, as well as resting and MEP measures, to help further clarify association specificity. Further, since all but one of the previous studies[19] were cross-sectional, future studies should employ prospective designs to elucidate static or dynamic pain sensitivity as predictors versus mediators of resting or MEP.

While not the primary focus of our study, findings for perceived health (also known as ‘self-rated health’) are notable since it’s a factor not commonly included in pain sensitivity studies. In fact, a basic literature search by our team revealed zero pain sensitivity studies in low back pain that reported perceived health effects. Still, poorer perceived health has been found to predict poorer outcomes among individuals with low back pain.[48–51] Worse, perceived health has been linked to hospitalizations and mortality.[52–54] It is not entirely clear why perceived health related to all pain measures in the current study, or why it was the strongest factor in all but one multivariate model. One explanation is that perceived health is a proxy of multimorbidity[55]; meaning that those with a greater number of health conditions (or more extensive health history) are more likely to have more clinical pain. While this possibility is limited in the current sample (since many comorbid health conditions were excluded based on eligibility criteria), poorer perceived health has been recently linked to multimorbidity among older adults.[56] An alternative explanation is that perceived health is a construct overlapping psychological distress. For example, perceived health was previously

associated with depression among individuals with persistent LBP.[57] Conceptually, perceived health may reflect a person's self-efficacy, i.e. poorer perceived health is indication of low confidence to confront pain and function. Given the strength of associations here and its established impact on health outcomes, perceived health should certainly be explored and expanded in LBP pain sensitivity studies.

Study limitations should be considered with our findings. First, since this is a secondary analysis, sample size estimation was based on the primary question aimed at comparing age groups.[21] Still, we were mindful of regression modeling practices including avoidance of multicollinearity and model overfitting. Second, as this study utilized a cross-sectional design, we were unable to test pain sensitivity as a risk factor for resting pain or movement evoked pain outcomes. Second, as this was a community-derived sample, these findings do not necessarily generalize to care-seeking patients with persistent LBP. Last, despite the inclusion of 90 participants, we cannot rule out the potential for more pain sensitivity measures being included in the multivariate models if a larger sample were analyzed.

To summarize, adults with persistent LBP demonstrated altered static and dynamic pain sensitivity when compared to age- and sex-matched controls. MEP may prove more relevant to pain sensitivity than resting or recalled pain based on the preponderance of bivariate associations; or alternatively, have specific associations to dynamic pain sensitivity (versus static pain sensitivity for resting pain). Future research will confirm such association specificity and also determine the extent to which pain sensitivity acts as a mediator of either resting pain or MEP.

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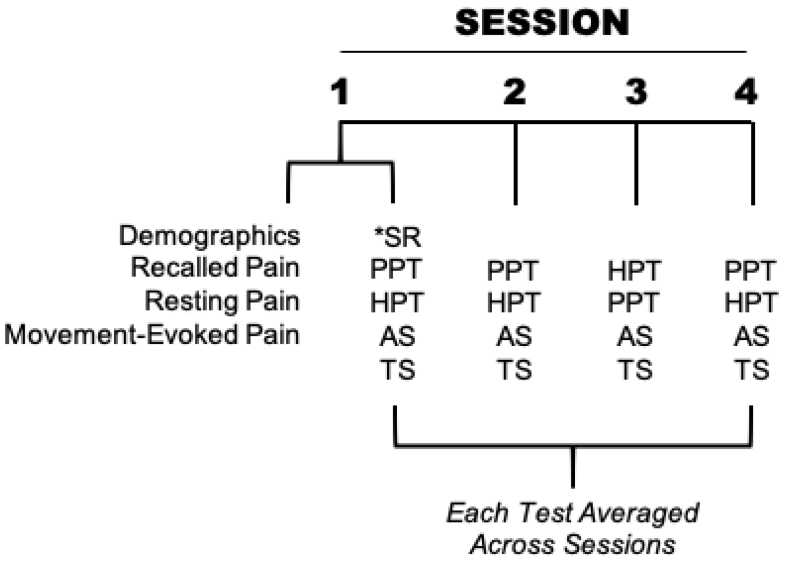

Figure 1). Study Procedures.

Participants completed four study sessions (up to 2 sessions per week with an average duration of 13.7 days and inter-session interval of 4.6 days), with clinical pain measures occurring in the first session before measuring pain sensitivity. Subsequent sessions included pain sensitivity, only. *SR=stimulus response ((*) = performed once to determine ramping temperature for aftersensations); PPT = Pressure Pain Threshold, performed at thenar eminence of right hand and bilateral PSIS of the low back; HPT = heat pain threshold; AS=Heat pain aftersensations; TS = Temporal summation of second heat pain.

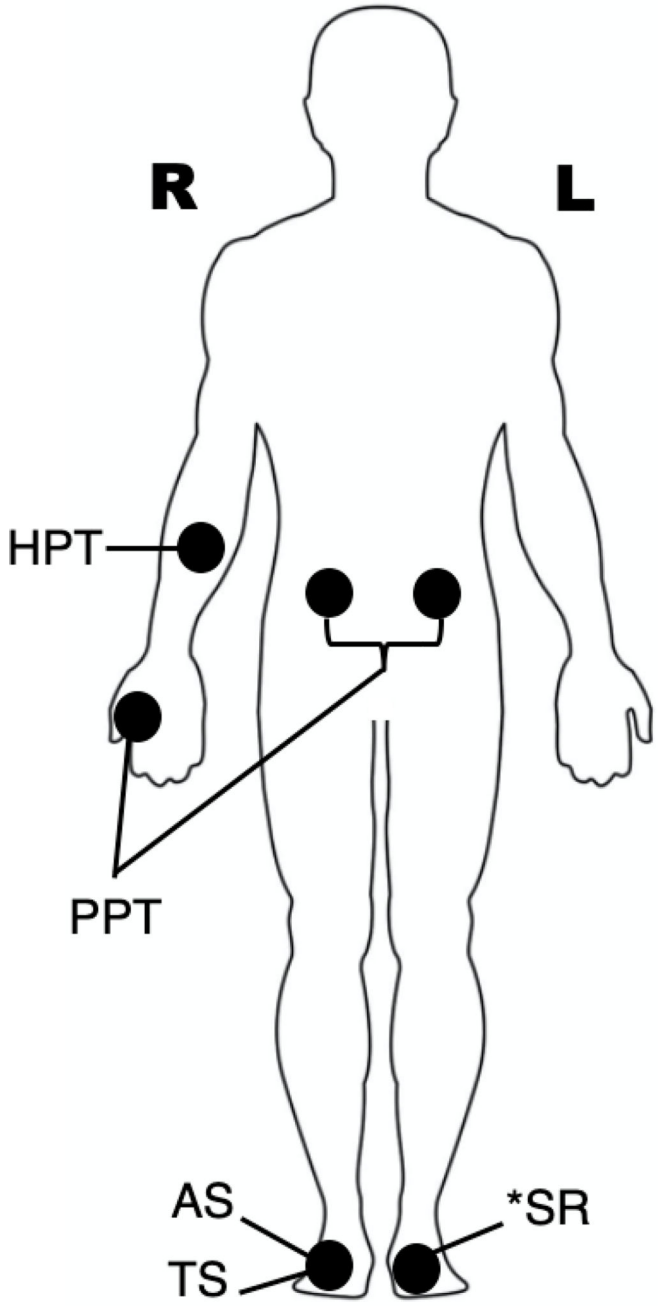

Figure 2). Pain Sensitivity Test Locations.

SR=stimulus response ((*)= performed once to determine ramping temperature for heat pain aftersensations); PPT = Pressure Pain Threshold, performed remotely at the right hand (thenar eminence) and locally at the low back (bilateral posterior superior iliac spines or PSIS); HPT= Heat pain threshold; AS = Heat pain aftersensations; TS= Temporal summation of second heat pain.

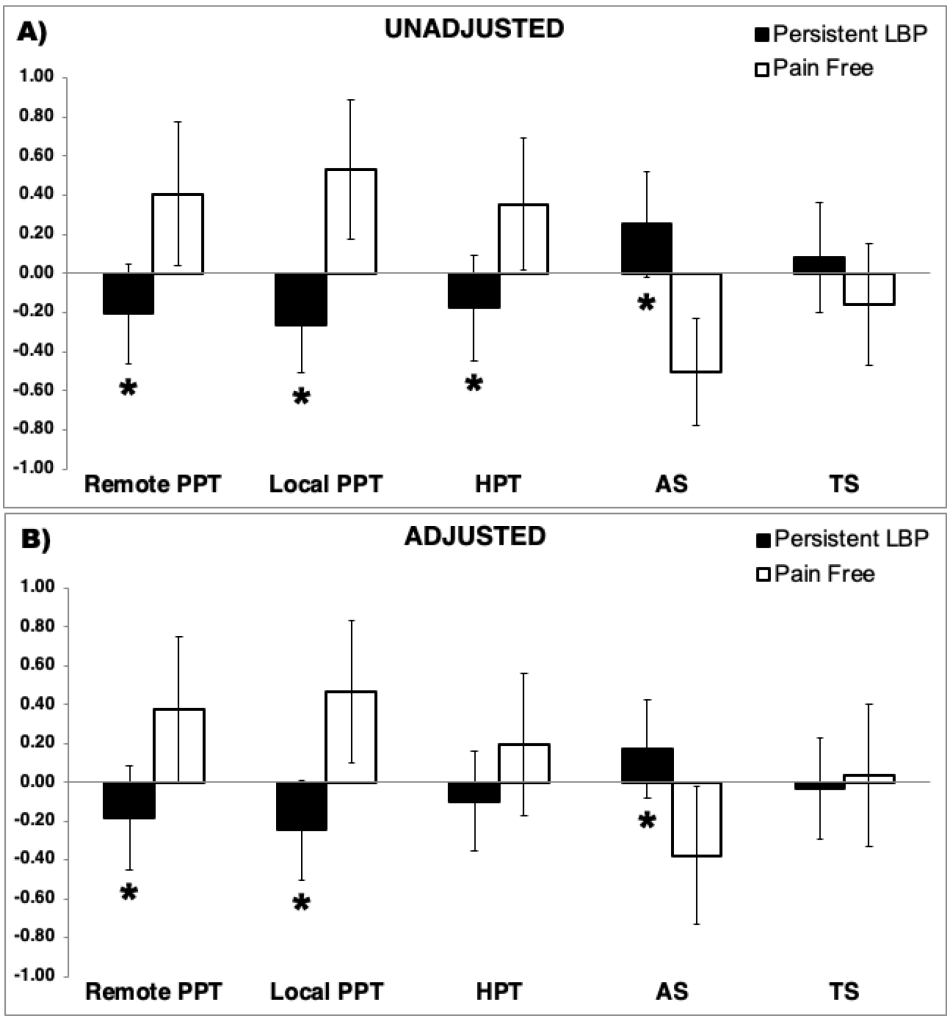

Figure 3). Mean Differences in Pain Sensitivity, Persistent LBP Versus Age- & Sex-Matched Controls (z-transformed).

(**A**) Unadjusted mean differences; (**B**) Mean differences after adjusting for education, global cognition, and perceived health; Remote PPT = pressure pain threshold hand (thenar eminence); Local PPT = pressure pain threshold low back (posterior superior iliac spine or PSIS); HPT = heat pain threshold; AS = Heat pain aftersensations; TS = Temporal summation of second heat pain. (*) = $p<.05$; Error bars= 95% confidence interval of the mean.

Table 1)

Demographic Characteristics, Persistent LBP vs. Pain-Free Controls

	CLBP (n=60)		PAIN FREE (n=30)		p
Age	47.67	(14.58)	47.57	(16.45)	0.977
Female (%)	68		63		0.635
Education (%)					
High School Attendance	38		17		**0.036**
College Attendance	62		83		
Global Cognition	27.70	(2.10)	28.60	(2.00)	0.060
Perceived Health (%)					
Excellent to Good	68		97		**0.002**
Fair to Poor	32		3		
Static Pain Sensitivity					
Pressure Pain Threshold - Thenar	5.00	(1.93)	6.20	(1.96)	**0.006**
Pressure Pain Threshold - PSIS	4.52	(2.32)	6.49	(2.33)	**<.001**
Heat Pain Threshold	41.74	(3.31)	43.47	(2.86)	**0.019**
Dynamic Pain Sensitivity					
Heat Pain Aftersensations	85.22	(61.61)	40.06	(43.11)	**<.001**
Temporal Summation of Second Heat Pain (TS)	2.51	(12.59)	−0.31	(9.56)	0.145
Clinical Outcome Measures					
Recalled Pain	4.98	(1.86)	0.20	(0.47)	**<.001**
Resting Pain	4.20	(2.46)	0.10	(0.31)	**<.001**
Movement-Evoked Pain	37.52	(25.00)	0.07	(0.27)	**<.001**

Table 2)

Bivariate Associations Between Demographics, Pain Sensitivity, & Clinical Pain Measures

A)	Remote PPT (Hand)		Local PPT (Low Back)		Heat Pain Threshold		Heat Pain Aftersensations		Temporal Summation (TS)	
	Rho	p	Rho	p	Rho	p	Rho	p	Rho	p
Demographics										
Age	0.021	0.877	**0.334**	**0.010**	0.076	0.569	0.091	0.498	0.162	0.226
Sex	−0.234	0.080	−0.199	0.135	0.041	0.762	0.067	0.618	0.048	0.719
Education	0.188	0.161	0.193	0.146	**0.298**	**0.023**	−0.076	0.569	−0.173	0.194
Global Cognition	0.183	0.176	0.207	0.122	0.205	0.126	−0.182	0.175	**−0.295**	**0.026**
Perceived Health	0.071	0.604	0.077	0.570	0.256	0.054	**−0.268**	**0.044**	**−0.349**	**0.008**

B)	Recalled Pain		Resting Pain		Movement-Evoked Pain	
	Rho	p	Rho	p	Rho	p
Demographics						
Age	0.024	0.855	−0.142	0.281	−0.142	0.279
Sex	0.180	0.168	0.107	0.414	−0.066	0.615
Education	**−0.357**	**0.005**	**−0.252**	**0.052**	**−0.232**	**0.075**
Global Cognition	**−0.289**	**0.026**	−0.086	0.516	**−0.244**	**0.062**
Perceived Health	**−0.321**	**0.013**	**−0.274**	**0.036**	**−0.445**	**<.001**
Pain Sensitivity						
Remote Pressure Pain Threshold (Hand)	−0.169	0.209	−0.194	0.149	−0.183	0.172
Local Pressure Pain Threshold (Low Back)	−0.204	0.125	**−0.291**	**0.027**	**−0.307**	**0.019**
Heat Pain Threshold	−0.161	0.228	−0.232	0.080	**−0.310**	**0.018**
Heat Pain Aftersensations	0.085	0.525	0.181	0.174	**0.361**	**0.005**
Temporal Summation (TS)	**0.351**	**0.007**	**0.436**	**0.001**	**0.488**	**<.001**

PPT= Pressure pain threshold; Temporal Summation (TS)= Temporal summation of second heat pain

Table 3)

Multivariate Associations with Recalled, Resting, and Movement-Evoked Pain

RECALLED PAIN

Temporal Summation of Second Heat Pain (TS)

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Education	**0.24**	**−0.229**	**(−0.461, −0.002)**
	Global Cognition		−0.153	(−0.370, 0.064)
	Perceived Health		**−0.246**	**(−0.483, −0.023)**
2	TS	0.00	0.029	(−0.181, 0.239)

RESTING PAIN

Local Pressure Pain Threshold (PPT Low Back)

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Age	**0.21**	−0.027	(−0.241, 0.186)
	Education		−0.193	(−0.420, 0.031)
	Global Cognition		−0.009	(−0.221, 0.204)
	Perceived Health		**−0.234**	**(−0.455, −0.025)**
2	PPT (Low Back)	**0.09**	**−0.319**	**(−0.523, −0.111)**

Temporal Summation of Second Heat Pain (TS)

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Education	**0.19**	−0.228	(−0.463, 0.005)
	Global Cognition		−0.011	(−0.232, 0.210)
	Perceived Health		**−0.231**	**(−0.471, 0.003)**
2	TS	0.01	0.125	(0.090, 0.339)

MOVEMENT-EVOKED PAIN

Local Pressure Pain Threshold (PPT Low Back)

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Age	**0.26**	−0.114	(−0.400, 0.173)
	Education		0.039	(−0.211, 0.280)
	Global Cognition		−0.147	(−0.399, 0.127)
	Perceived Health		**−0.433**	**(−0.598, −0.136)**
2	PPT (Low Back)	0.03	−0.179	(−0.452, 0.095)

Heat Pain Threshold (HPT)

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Education	**0.22**	0.055	(−0.196, 0.293)
	Global Cognition		−0.115	(−0.347, 0.135)
	Perceived Health		**−0.374**	**(−0.548, −0.085)**
2	HPT	0.05	−0.240	(−0.450, 0.014)

Heat Pain Aftersensations

			Final Model	
Bk	***Factor***	***$R^2\Delta$***	***Beta***	***95% CI Beta***
1	Education	**0.22**	0.001	(−0.233, 0.235)
	Global Cognition		−0.098	(−0.324, 0.143)
	Perceived Health		**−0.331**	**(−0.507, −0.053)**
2	Aftersensations	**0.10**	**0.328**	**(0.076, 0.516)**

Temporal Summation of Second Heat Pain (TS)

RECALLED PAIN	RESTING PAIN	MOVEMENT-EVOKED PAIN			
				Final Model	
		Bk	*Factor*	$R^2\Delta$	*Beta*	*95% CI Beta*
			Education		0.011	(−0.220, 0.240)
		1	Global Cognition	**0.22**	−0.047	(−0.279, 0.191)
			Perceived Health		**−0.286**	**(−0.471, −0.013)**
		2	TS	**0.12**	**0.389**	**(0.112, 0.552)**

Bk=Block; $R^2\Delta$= R^2 change with addition of block; Final Model = Comparison of standardized regression coefficients (beta) and beta 95% confidence intervals for all factors in the final model.