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# Spine Kinematics and Muscle Activities in Non-specific Chronic Low Back Pain Subgroups in Sitting

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in conjunction with the
Faculty of Graduate Studies

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# Spine Kinematics and Muscle Activities in Non-specific Chronic Low Back Pain Subgroups in Sitting

by

Mansoor Ahmed Alameri

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A Dissertation submitted in partial satisfaction of
the requirements for the degree
Doctor of Philosophy in Physical Therapy

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December 2019

Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

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# ABBREVIATIONS

| | |
|---|---|
| NSCLBP | Non-Specific Chronic Low Back Pain |
| MCI | Motor Control Impairment |
| FP | Flexion Pattern |
| AEP | Active Extension Pattern |
| ST | Sacral Tilt |
| L3 | Third Lumbar Vertebrae |
| RLLA | Relative Lower Lumbar Angle |
| LBP | Low Back Pain |
| CLBP | Chronic LBP |
| MDCS | Multidimensional Classification System |
| EMG | Electromyography |
| MVIC | Maximal Voluntary Isometric Contraction |
| sMVIC | Submaximal Voluntary Isometric Contraction |
| CCIS | Co-contraction Indices |
| LLUH | Loma Linda University health |
| BMI | Body Mass Index |
| NPRS | Numeric Pain Rating Scale |
| RMDQ | Ronald Morris Disability Questionnaire |
| TSK | Tampa Scale of Kinesophobia |
| 2D | 2-Dimensional |
| SD | Standard Deviation |
| ANOVA | Analysis of Variance |

ABSTRACT OF THE DISSERTATION

Spine Kinematics and Muscle Activities in Non-specific Chronic Low Back Pain Subgroups in Sitting

by

Mansoor Ahmed Alameri

Doctor of Philosophy, Graduate Program in Physical Therapy
Loma Linda University, December 2019
Dr. Everett Lohman III, Chairperson

**Background:** Although, non-specific chronic low back pain (NSCLBP) has been associated with motor control impairments, little is known about the possible driving mechanisms of pain development overtime during prolonged sitting period. Therefore, the purpose of this study was to examine the differences in lumbosacral postures and muscle activities in adults with and without NSCLBP, and their role on pain development during a 1-hour of prolonged sitting task.

**Methods:** Twenty NSCLBP subjects with motor control impairment (MCI) [10 classified as having flexion pattern (FP) disorder, and 10 with active extension pattern (AEP) disorder], and 10 healthy controls participated in the study. Subjects underwent a 1-hour sitting protocol on a standard office chair. Lumbosacral postures including sacral tilt (ST), third lumbar vertebrae (L3) position, and relative lower lumbar angle (RLLA) were recorded using a two-dimensional inclinometer. In addition, four trunk muscle activities including amplitudes and co-contractions were recorded using electromyography over the 1-hour period. Perceived back pain intensity was recorded using a numeric pain rating scale every 10 minutes throughout the sitting period.

**Results:** All study groups presented with significantly distinctive lumbosacral kinematics at the lowest level of pain (the beginning of the sitting period) ($p<0.05$), as well as at the highest level of pain (the end of the sitting period) ($p\leq0.05$). The MCI subgroups showed a significant deterioration in lumbosacral kinematics and pain levels overtime ($p<0.01$). The directions of deterioration in lumbosacral kinematics over the 1-hour sitting period occurred in the direction of the motor control impairment (kyphosis for FP subgroup or lordosis for the AEP subgroup). Both MCI subgroups reported a similarly significant increase in pain through mid-sitting ($p<0.001$). However, after mid-sitting, the AEP subgroup displayed a significantly reversed decrease in the lordotic postures ($p=0.001$) which was accompanied by much less increase in pain level compared to the FP subgroup. No significantly distinctive trunk muscles' activities were found at the beginning of sitting ($p>0.05$), nor did the muscle activities change overtime.

**Conclusion**: The present study's findings suggest that MCI subgroups presented with distinctive underlying maladaptive postural patterns. However, the significant increase in pain over the 1-hour sitting might not be only attributed to the inherent maladaptive postures, also it may be related to the directional deterioration in lumbosacral postures overtime.

**Keywords**: Low back pain, Motor control impairment, Flexion pattern disorder, Active extension pattern disorder, Prolonged sitting, Lumbosacral kinematics, Muscle Activity.

# CHAPTER ONE

# INTRODUCTION

## Low Back Pain

Low back pain (LBP) is a common health condition associated with physical, social and economic burden, and it has been identified as the leading cause of disability globally (1, 2). Its prevalence per month is estimated to be 23% in general population and continue to exponentially rise (3). Although, most LBP cases recover within 3 to 4 weeks (4-6), a quarter to a third continue to report pain which becomes chronic (6-8). Previous research reported that approximately 85% of chronic LBP (CLBP) conditions have unrecognizable cause or specific pathology and are often identified as non-specific CLBP (NSCLBP) disorder (6, 7). The inability to define an underlying cause of NSCLBP disorder has been primarily attributed to the heterogeneity, multidimensionality, and complexity of the disorder (9, 10). Thus, defining homogenous subgroups as well as considering a broad biopsychosocial model of this pain disorder have been ranked as a top priority in spine pain management research (9, 11-14).

### *Classification of Low Back pain*

A multidimensional classification system (MDCS) has been proposed to classify NSCLBP disorders based on biomechanical and psychosocial constructs of the disorder in an attempt to understand the possible underlying mechanisms of pain (9, 11-13, 15, 16). This system has been widely accepted in literature (9), but most importantly, treatment approaches based on this system have been shown to be effective in reducing back pain (9, 14, 17). In this system, a large number of NSCLBP patients are classified as

motor control impairment (MCI) subgroups, in which a maladaptive motor response is reported as the main possible driver of pain (9). This motor dysfunction is thought to be secondary to the loss of motor control of the moving segment around the neutral zone resulting in a non-physiological spinal loading or movement (9, 11, 13, 15).

### *Lumbosacral Kinematics in Low Back Pain*

Previous researches have reported that individuals with NSCLBP with MCI exhibited high levels of fear avoidance behavior, abnormal activation of trunk muscles (13, 16), altered spinal position sense (18), and assumed spinal end ranges toward the direction of pain provocation - commonly flexion [called flexion pattern subgroup (FP)] or extension [called active extension pattern subgroup (AEP)] (17, 19). Previous studies reported that during 5-10 seconds sitting, patients with FP subgroup assumed an end-range flexion position (kyphosis) of the lumbar spine, whereby, AEP patients actively postured themselves into lordosis and showed high levels of activation of trunk muscles (13, 15). Although the suggested postural faults, in the studies cited above, were inherent and displayed prior to the actual onset of pain (9, 11, 13, 15), they are thought to predispose one to pain development over time. Therefore, studies examining how MCI subgroups operate over an extended period of time might be needed to confirm this notion.

In an attempt to address the postural behaviors inherently adopted by the MCI subgroups, O'Keeffe, Dankaerts (17), (20) examined the effect of altering sitting kinematics using chairs with adjustable seatpan inclinations on back discomfort levels. Results of both studies showed that the level of discomfort significantly decreased during

sitting on a standardized chair for the AEP subgroup, and during sitting on a forward-inclined chair for the FP subgroup (17). Although these findings are viable means for providing answers regarding pain development over time, no study has yet examined the postural behavioral patterns of the lumbosacral region and their influence on pain during the exposure to prolonged tasks such as siting. Therefore, studies examining lumbosacral kinematics at lowest and highest levels of pain provoked by means of prolonged sitting are needed to further understand the nature of the relationship between lumbosacral kinematics and pain development over time among the MCI subgroups.

### *Prolonged Sitting and Low Back Pain*

Prolonged sitting is widely accepted as a risk factor in developing LBP (21), and frequently reported to aggravate pain in the NSCLBP population (17, 19, 22). Because the prevalence of occupations that requires sitting for an extended period of time increases (13, 15, 16, 23), research examining prolonged sitting posture in homogenous NSCLBP patients might be relevant. Identifying distinctive postural patterns among the NSCLBP subgroups in presence of pain, using the validated MDCS, will facilitate further understanding of the mechanism of pain development, and eventually support the ability of clinicians to provide customized, subgroup-specific interventions to optimize outcomes. Furthermore, because MCI subgroups reported pain during the exposure to prolonged tasks, the present study focused on the MCI subgroups with either FP or AEP disorder.

### *Lumbosacral Muscle Activation in Low Back Pain*

Abnormal neuromuscular control and its contribution to pain development has been well documented in patients with NSCLBP [9, 10]. Despite the considerable amount of evidence suggesting the presence of motor control faults in NSCLBP patients, the nature of these faulty patterns in response to pain provocation are highly inconsistent [11-15]. Several research studies reported no differences in trunk muscle activations in this population [11, 14, 15]. In contrast, one study reported a decrease in muscle activations in patients with NSCLBP [16] while others reported an increase in muscle activities [10, 17]. These inconsistent findings were commonly attributed to the "washout effect" when interpreting EMG data of heterogeneous CLBP patients [12]. Therefore, a Multidimensional Classification system (MDCS) emerged in which, a large number of those patients was classified based on the underlying mechanical basis of their pain disorder [9, 11, 12, 18-20].

A study that used the MDCS, found that the levels of activity of lumbar muscles were higher in extension-related NSCLBP, namely active extension pattern (AEP) subgroup, when compared to healthy subjects and flexion-related back pain, called flexion pattern (FP) subgroup during 5-second sitting [12]. This increase in back muscle activity is thought to predispose those patients to pain [9, 12]. Contrary to that, muscular patterns of NSCLBP patients were found highly variable in other research studies using the MDCS [11, 14, 15]. Most of these studies examined the neuromuscular functions of the trunk muscles using activation amplitudes as an indicator for the motor response [11-15]. However, a muscle pairing analysis (an analysis of localized co-contraction) was not considered. This type of analysis allows a better understanding of the activation pattern of

one abdominal and another back muscle at the time of a specific activation, which allows to thoroughly understand the relationships between these two paired muscles during known tasks [17, 21].

So far, research data showed inconsistent differences in trunk muscle amplitudes among MCI subgroups; however, co-activation analysis has not been previously performed for the MCI subgroups. Therefore, identifying co-contraction patterns and understanding the relationship between abdominal and back muscles among the MCI subgroups in presence of pain provocative would enable clinicians to provide customized, subgroup-specific interventions based on the underlying motor control impairments. Furthermore, patients with MCI, FP and AEP subgroups, demonstrate pain provocation during the exposure to static loading such that occurring during prolonged sitting [9]. Because prolonged sitting is frequently reported to aggravate pain in NSCLBP population [17, 18], research examining sitting nature is becoming increasingly relevant.

Therefore, the purpose of this study was to examine whether the trunk muscles' co-contraction a) differed among FP, AEP subgroups and healthy controls, and b) changed overtime when pain is at its lowest and at its highest over a 1-hour sitting task. It was hypothesized that subjects with FP and AEP disorders would display higher coactivation patterns as the pain is at its lowest level when compared to healthy controls, and that these patterns would increase toward the end of sitting period as the pain is at its highest level. Additionally, the second purpose of this study was to examine whether the lumbosacral spine postures a) differed among FP, AEP subgroups and healthy controls, b) changed overtime when pain is at its lowest and at its highest over a 1-hour sitting task. It was hypothesized that subjects with FP disorder would display lumbosacral

kyphotic postures whereas those with AEP would assume lumbosacral lordosis as the pain is at its lowest level, and that these postures would deteriorate toward the end ranges over the 1-hour sitting period as the pain is at its highest level.

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# CHAPTER TWO

# COMPARISONS OF LUMBOSACRAL KINEMATICS DURING PROLONGED SITTING IN NON-SPECIFIC CHRONIC LOW BACK PAIN SUBGROUPS; A CROSS-SECTIONAL STUDY


## Abstract

### *Background*

Although, non-specific chronic low back pain (NSCLBP) has been associated with abnormal lumbosacral kinematics, little is known about the possible driving mechanisms of pain development overtime during prolonged sitting period. Therefore, the purpose of this study was to examine the differences in lumbosacral postures in adults with and without NSCLBP, and their role on pain development during a 1-hour of prolonged sitting task.

### *Methods*

Twenty NSCLBP subjects with motor control impairment (MCI) [10 classified as having flexion pattern (FP) disorder, and 10 with active extension pattern (AEP) disorder], and 10 healthy controls participated in the study. Subjects underwent a 1-hour sitting protocol on a standard office chair. Lumbosacral postures including sacral tilt (ST), third lumbar vertebrae (L3) position, and relative lower lumbar angle (RLLA) were recorded using a two-dimensional inclinometer over the 1-hour period. Perceived back pain intensity was recorded using a numeric pain rating scale every 10 minutes throughout the sitting period.

### *Results*

All study groups presented with significantly distinctive lumbosacral kinematics at the lowest level of pain (the beginning of the sitting period) ($p<0.05$), as well as at the

highest level of pain (the end of the sitting period) ($p \leq 0.05$). The MCI subgroups showed a significant deterioration in lumbosacral kinematics and pain levels overtime ($p < 0.01$). The directions of deterioration in lumbosacral kinematics over the 1-hour sitting period occurred in the direction of the motor control impairment (kyphosis for FP subgroup or lordosis for the AEP subgroup). Both MCI subgroups reported a similarly significant increase in pain through mid-sitting ($p < 0.001$). However, after mid-sitting, the AEP subgroup displayed a significantly reversed decrease in the lordotic postures ($p = 0.001$) which was accompanied by much less increase in pain level compared to the FP subgroup.

***Conclusion***

The present study's findings suggest that MCI subgroups presented with distinctive underlying maladaptive postural patterns. However, the significant increase in pain over the 1-hour sitting might not be only attributed to the inherent maladaptive postures, also it may be related to the directional deterioration in lumbosacral postures overtime.

***Keywords***

Low back pain, Motor control impairment, Flexion pattern disorder, Active extension pattern disorder, Prolonged sitting, Lumbosacral kinematics.

## Introduction

Low back pain (LBP) is a common health condition associated with physical, social and economic burden, and it has been identified as the leading cause of disability globally (1, 2). Its prevalence per month is estimated to be 23% in general population and continue to exponentially rise (3). Although, most LBP cases recover within 3 to 4 weeks (4-6), a quarter to a third continue to report pain which becomes chronic (6-8). Previous research reported that approximately 85% of chronic LBP (CLBP) conditions have unrecognizable cause or specific pathology and are often identified as non-specific CLBP (NSCLBP) disorder (6, 7). The inability to define an underlying cause of NSCLBP disorder has been primarily attributed to the heterogeneity, multidimensionality, and complexity of the disorder (9, 10). Thus, defining homogenous subgroups as well as considering a broad biopsychosocial model of this pain disorder have been ranked as a top priority in spine pain management research (9, 11-14).

A multidimensional classification system (MDCS) has been proposed to classify NSCLBP disorders based on biomechanical and psychosocial constructs of the disorder in an attempt to understand the possible underlying mechanisms of pain (9, 11-13, 15, 16). This system has been widely accepted in literature (9), but most importantly, treatment approaches based on this system have been shown to be effective in reducing back pain (9, 14, 17). In this system, a large number of NSCLBP patients are classified as motor control impairment (MCI) subgroups, in which a maladaptive motor response is reported as the main possible driver of pain (9). This motor dysfunction is thought to be secondary to the loss of motor control of the moving segment around the neutral zone resulting in a non-physiological spinal loading or movement (9, 11, 13, 15).

Previous researches have reported that individuals with NSCLBP with MCI exhibited high levels of fear avoidance behavior, abnormal activation of trunk muscles (13, 16), altered spinal position sense (18), and assumed spinal end ranges toward the direction of pain provocation - commonly flexion [called flexion pattern subgroup (FP)] or extension [called active extension pattern subgroup (AEP)] (17, 19). Previous studies reported that during 5-10 seconds sitting, patients with FP subgroup assumed an end-range flexion position (kyphosis) of the lumbar spine, whereby, AEP patients actively postured themselves into lordosis and showed high levels of activation of trunk muscles (13, 15). Although the suggested postural faults, in the studies cited above, were inherent and displayed prior to the actual onset of pain (9, 11, 13, 15), they are thought to predispose one to pain development over time. Therefore, studies examining how MCI subgroups operate over an extended period of time might be needed to confirm this notion.

In an attempt to address the postural behaviors inherently adopted by the MCI subgroups, O'Keeffe, Dankaerts (17), (20) examined the effect of altering sitting kinematics using chairs with adjustable seatpan inclinations on back discomfort levels. Results of both studies showed that the level of discomfort significantly decreased during sitting on a standardized chair for the AEP subgroup, and during sitting on a forward-inclined chair for the FP subgroup (17). Although these findings are viable means for providing answers regarding pain development over time, no study has yet examined the postural behavioral patterns of the lumbosacral region and their influence on pain during the exposure to prolonged tasks such as siting. Therefore, studies examining lumbosacral kinematics at lowest and highest levels of pain provoked by means of prolonged sitting

are needed to further understand the nature of the relationship between lumbosacral kinematics and pain development over time among the MCI subgroups.

Prolonged sitting is widely accepted as a risk factor in developing LBP (21), and frequently reported to aggravate pain in the NSCLBP population (17, 19, 22). Because the prevalence of occupations that requires sitting for an extended period of time increases (13, 15, 16, 23), research examining prolonged sitting posture in homogenous NSCLBP patients might be relevant. Identifying distinctive postural patterns among the NSCLBP subgroups in presence of pain, using the validated MDCS, will facilitate further understanding of the mechanism of pain development, and eventually support the ability of clinicians to provide customized, subgroup-specific interventions to optimize outcomes. Furthermore, because MCI subgroups reported pain during the exposure to prolonged tasks, the present study focused on the MCI subgroups with either FP or AEP disorder. Therefore, the purpose of this study was to examine whether the lumbosacral spine postures a) differed among FP, AEP subgroups and healthy controls, b) changed overtime when pain is at its lowest and at its highest over a 1-hour sitting task. It was hypothesized that subjects with FP disorder would display lumbosacral kyphotic postures whereas those with AEP would assume lumbosacral lordosis as the pain is at its lowest level, and that these postures would deteriorate toward the end ranges over the 1-hour sitting period as the pain is at its highest level.

## Materials and Methods

### *Study's aim, design and sitting*

The aim of this cross-sectional study was to examine the differences and overtime changes in lumbosacral postures in adults with and without NSCLBP, and their role on

pain development during a 1-hour of prolonged sitting task at a work-simulated laboratory setting.

### *Participants*

A total of 38 subjects; 27 NSCLBP with MCI and 11 healthy controls were recruited from private outpatient physical therapy clinics and Loma Linda University Health (LLUH). Because of the strict inclusion criteria, 5 NSCLBP subjects were excluded due to low pain level in the NPRS (<2/10); 2 NSCLBP subjects due to the inability to establish mechanical basis of the disorder, and 1 healthy control was also excluded due to pain development after 40 minutes of sitting. The recruitment of NSCLBP subjects was completed by two therapists (MA and AS) independently via a comprehensive subjective assessment and physical examination described elsewhere (9, 15, 24). Only subjects with FP or AEP, in which both therapists were in agreement, were included in the study. Previous research reported a substantial agreement between therapists upon the classification of NSCLBP with MCI advocating its intra-rater reliability (25, 26). Inclusion and exclusion criteria are summarized in **Table 1.**

**Table 1.** Inclusion and Exclusion Criteria

| Inclusion Criteria | Exclusion Criteria |
|---|---|
| • ≥ 3 months NSCLBP<br>• ≥ 5 points scored on RMDQ score<br>• Pain in the lower lumbosacral region<br>• Absence of "red flags" (such as inflammatory diseases or causa equina)<br>• Absence of dominant "yellow flags" (such as identification of beliefs, emotions, and behaviors that interact with the pain problem)<br>• Clear mechanical basis of disorder<br>• Associated impairments in the control of the motion segment(s) in the provocative movement direction (s)<br>• Absence of impaired movement of the symptomatic segment in the painful direction of movement (based on clinical joint mobility examination)<br>• Diagnosis of an FP or AEP disorder (both examining clinicians independently agreed upon the diagnosis) | • <5 points scored on RMDQ score<br>• Signs of neurologic involvement, e.g., radicular pain, and more generalized pain<br>• Evidence of specific diagnosis, e.g., spondylolisthesis, inflammatory disease,<br>• Previous spine surgery<br>• Pregnant at the time of the study or 6 months postpartum |

**Abbreviation:**
NSCLBP, non-specific chronic low back pain; RMDQ, Ronald Morris Disability Questionnaire; LBP, low back pain; FP, flexion pattern; AEP, active extension pattern.

Ethical approval was obtained from the Institutional Review Board at LLUH #5180306. Since subjects often sit for prolonged periods and reported LBP during performing this, they were informed that they were likely to experience LBP during a 1-

hour sitting period. However, they were informed that they could discontinue the testing protocol at any moment if they wished. All subjects read and signed a written informed consent prior to participation in the study.

Gender, age, anthropometric data [weight, height, and body mass index (BMI)], perceived pain using the Numeric Pain Rating Scale (NPRS) [in the past week, 24 hours and at baseline] (19, 23, 27), pain duration, pain-related disability [Ronald Morris Disability Questionnaire (RMDQ) (28)] and Tampa Scale of Kinesophobia (TSK) (29)] were compared by group in **Table 2.** Data collection were conducted at the Orthopedic and Manual Therapy Laboratory, Department of Physical Therapy, LLUH, California, United States.

### *Instrumentation*

**2-Dimensional Inclinometer (2D-Inclinmeter)**

2D inclinometer sensors [4 × 2.5 × 1.4 cm x 45.5 g] (Noraxon USA, Inc, Scottsdale, AZ) were used to record lumbosacral angles during the 1-hour siting period. 2D inclinometer is a noninvasive electromagnetic device, which measures the tilt level of the sensor to the ground in two planes expressed in degrees (30, 31). In alignment with previous studies, the postures the sacral tilt and third lumbar vertebrae (L3) were recorded. Also, the sum of the sacral tilt and its correspondent L3 position was manually

**Table 2.** Mean (SD) of Baseline Characteristics by Study Group (N = 30).

| | **FP (n-10)** | **AEP (n=10)** | **CG (n=10)** | **p-value** |
|---|---|---|---|---|
| **Male (n)** | 7 | 4 | 6 | 0.39 |
| **Age, y** | 27.8 (4.0) | 27.9 (5.3) | 27 (5.8) | 0.91 |
| **Height, feet** | 5.8 (0.3) | 5.3 (0.3) | 5.2 (0.5) | 0.06 |
| **Mass, lbs** | 157.5 (30.3) | 154.4 (36.1) | 143.8 (25.0) | 0.58 |
| **BMI ($kg/m^2$)** | 24.8 (4.5) | 25.0 (4.4) | 25.2 (2.9) | 0.98 |
| **NPRS (average/wk/100mm)** | 45.3 (14.1) | 40.0 (19.2) | - | 0.50 |
| **NPRS (average/24hr/100mm)** | 19.7 (13.8) | 12.0 (10.9) | - | 0.31 |
| **NPRS (average/Baseline)** | 17.8 (10.1) | 7.9 (6.9) | - | 0.02 |
| **Pain Duration, y** | 3.5 (5.3) | 6.0 (5.6) | - | 0.33 |
| **RMDI (%)** | 7.2 (2.2) | 6.0 (1.5) | - | 0.24 |
| **TSK (64 score)** | 14.2 (5.7) | 22.8 (8.4) | - | 0.06 |

**Abbreviation:** SD, Standard Deviation; FP, Flexion Pattern; AEP, Active Extension Pattern; CG, Control Group; BMI, Body Mass Index; NPRS, Numeric Pain Rating Scale; RMDI, Ronald Morris Disability Index; TSK, Tampa Scale of Kinesophobia

calculated to indicate the global position of the lumbosacral region, namely as the relative lower lumbar angle (RLLA) (13, 15).

### Perceived Pain

While it is challenging to measure pain intensity (19), perceived pain scores was measured using the open NPRS during the 1-hour sitting period. The NPRS consists of a 100 mm horizontal line, anchored by the descriptors "no pain" and "worst pain imaginable" (27).

## *Procedures*

### Placement of the 2D-Inclinometer Sensors

A pair of 2D-sensor was attached using double-sided tape to continuously measure the sacral tilt, L3 angles throughout the 1-hour sitting period. The therapist identified anatomical landmarks for each subject and positioned the sensors on the landmarks. Specifically, one sensor was placed over the spinous process of S2 while the other sensor was placed over the spinous process of L3. The same therapist positioned all sensors to ensure consistency.

### Pain Measurement

Perceived pain was collected immediately prior to the beginning of the sitting protocol, and every 10 minutes throughout the 1-hour. Thus, a total of seven readings for each subject was taken. All subjects were asked to rate their pain by making a vertical line in the open NPRS at the point corresponding to their level of pain/discomfort. To

avoid artificial increase in the NPRS scores, all subjects were asked to focus on pain intensity rather than the location of their pain (19, 23). Additionally, subjects were allowed to compare their current NPRS score with the preceding scores to minimize unintended rating variations when drawing lines correspondent to their pain (19).

**1-Hour Sitting Protocol**

Following sensor placements, all subjects underwent a 1-hour sitting protocol in which they sat on an office chair reading pre-selected passages. Prior to sitting, the chair was modified by removing the backrests and armrests so they do not interfere with data collection (23). Although, this might alter the sitting behavior of the subjects, previous study found no difference in back discomfort levels when sitting in an office chair with backrest or without backrest (20). Therefore, this modification is deemed to be appropriate. In addition, the height of the chair was adjusted so that the subjects sat with hips and knees approximately at $90^0$ of flexion (23) and feet rested on the floor (17). Subjects were then provided with a standard office workstation setup, including a monitor, with the top of the screen at eye level, a keyboard and a mouse (23). Subjects were instructed to read and follow the text on the monitor with the mouse cursor using the right hand and pressing the 'Shift' key on the keyboard to move to the next paragraph using the left hand (23, 32). The subjects' elbows were kept at $90–100^o$ of flexion while reading, thus the height of the keyboard and mouse was adjusted to maintain this elbow angle (17). The distance from the keyboard was standardized for all subjects, in which the edge of the keyboard was in line with the radial styloid process and a distance of approximately 30 cm to subjects' greater trochanter (17). Thus, the potential for

confounding variables to effect the study findings was minimized. Finally, just prior to launching the 1-hour sitting protocol, all subjects were instructed to "sit as they normally will' on their office chairs.

Over the 1-hour sitting period, the degree of tilt of the sacrum and lumbar spine (15, 30) were recorded. Pain levels (19, 23) were also recorded at baseline and every 10 minutes throughout the sitting protocol.

### *Data Processing*

#### Sacral Tilt, Lumber 3 Position (L3), Relative Lower Lumbar Angles (RLLA)

For the sacral tilt and L3 angle in relation to the ground, the angles were recorded for 45 seconds before the 1-hour sitting period and the mean recorded angle was selected as reference value and used for calibration (31). To measure the deviation of each sensor from sagittal plane, the sacral tilt and L3 angle were measured throughout the entire 1-hour sitting period and every 10 minutes then normalized by subtracting the mean tilt/angle from its reference value, expressed in degrees and used for analysis (15). For the sacral tilt, a positive value indicates an anterior sacral tilt while a negative value indicates a posterior sacral tilt. For the L3 angle, a positive value indicates an extension whereas a negative value indicates a flexion. Furthermore, the sum of calibrated sacral tilt and its correspondent L3 angle was used to manually calculate the RLLA over the entire 1-hour sitting period and every 10 minutes. The RLLA represents the position of pelvis in relation to lower lumbar spine and is formed from the intersection between the inclination of the sensors lines at L3 and S2 (15). A positive value of the RLLA indicates a lordosis and a negative value indicates a lower lumbar kyphosis. The mean sacral tilt, L3 and

RLLA were selected over the entire 1-hour sitting, as well as at the beginning of the sitting period (baseline) and every 10 minutes throughout the 1-hour testing period (a total of 7 values) for analysis.

**Numeric Pain Rating Scale (NPRS)**

Post-collection, the perceived pain (a total of 7 readings) was used for analysis. For the pain subgroups, the NPRS scores were used to determine which data to be used for the primary analysis for each subject. For example, if a subject reported the lowest level of pain at the beginning, and the highest pain level at the third 10-minute interval of the sitting period, then only lumbosacral angles at the beginning of the sitting period and third 10-minute intervals were used for comparison within the same group and between study's groups. This way, we were able to compare the lumbosacral postures of each subject when the pain was at its lowest and highest levels.

## Statistical analysis

Data was summarized using mean and standard deviation (SD) for quantitative variables and counts (%) for qualitative variables. The normality of continuous variables was examined using Shapiro Wilk's test and Q-Q normality plots. The distribution of the subjects' characteristics by study group were evaluated using chi-square for gender, one-way Analysis of Variance (ANOVA) for age, height, mass and BMI, and independent t-test for duration of pain, NPRS (during past 24 hours, past week, and baseline), TSK and RMDI scores.

The primary analysis included a comparison of lumbopelvic kinematics (sacral tilt, L3 and RLLA) across groups at the lowest (baseline) and highest level of pain (minute 60) using one-way ANOVA (with post-hoc Bonferroni if results were significant). The secondary analysis included a comparison of lumbopelvic kinematics across groups over the entire 1-hour sitting using one-way ANOVA (with post-hoc Bonferroni). A third analysis included a 3x7 mixed factorial ANOVA (between factor: group; within factor: time) to examine changes in lumbopelvic kinematics and NPRS by study group over time. If the group x time interaction effect in the mixed factorial ANOVA was statistically significant, change from baseline was compared among groups at each time period (total of six "10-minute intervals") using one-way ANOVA (with post-hoc Bonferroni). If the interaction was not statistically significant, the between-groups comparison was considered not statistically significant. However, if the main effect of time was significant in the mixed factorial ANOVA, a one-way repeated measures ANOVA (with post-hoc Bonferroni) was used to examine changes over time within-groups separately. The level of significance was set at $p \leq 0.05$. Statistical analysis was performed using IBM SPSS Software version 24 for Windows (Chicago, IL, USA).

### *Sample size estimate*

For the primary and secondary analyses, a sample size of 30 subjects was estimated using a large effect size ($\eta^2=0.26$), level of significance ($\alpha= 0.05$), and power of 0.80. For the third analysis, a sample size of 30 subjects was estimated using a moderate effect size for the group x time interaction (partial $\eta^2=0.06$), level of significance ($\alpha= 0.05$), and power of 0.90.

## Results

A sample of 30 subjects with mean age 27.6±4.9 years, mass151.9±30.3 lbs., height 5.4±0.4 feet, BMI 25.0 ±3.9 kg/m2 participated in this study. Fifty-seven percent of the subjects were males (n=17). The distribution of all quantitative variables was approximately normal. There was no significant difference in subjects' characteristics by study group ($p>0.05$). Subjects' characteristics are summarized in **Table 2.**

### *Primary Analysis*

**Figure 1.** shows the differences in lumbosacral kinematics between groups at the beginning and the end of 1-hour sitting. There was a significant difference in ST and L3 angle among the three study groups at the lowest level of pain, which was at beginning of the sitting period, ($p=0.029$, $\eta 2= 0.23$ for ST and $p<0.001$, $\eta 2= 0.44$ for L3 angle). Bonferroni post hoc comparisons showed that the difference in ST was only significant between the FP and AEP subgroups ($p=0.031$), namely, the FP subgroup had slight posterior sacral tilt (-0.45°±2.02°), while the AEP subgroup had an increased anterior sacral tilt (3.35°±2.91°). In addition, the difference in L3 angle was only significant between FP subgroup and healthy controls ($p=0.004$), and between FP and AEP subgroups ($p=0.001$). Specifically, FP subgroup had greater L3 flexion (-3.11°±2.53°) compared to healthy controls (0.53°±2.48°) and AEP subgroup (1.19°±1.64°) who demonstrated slight L3 extension.

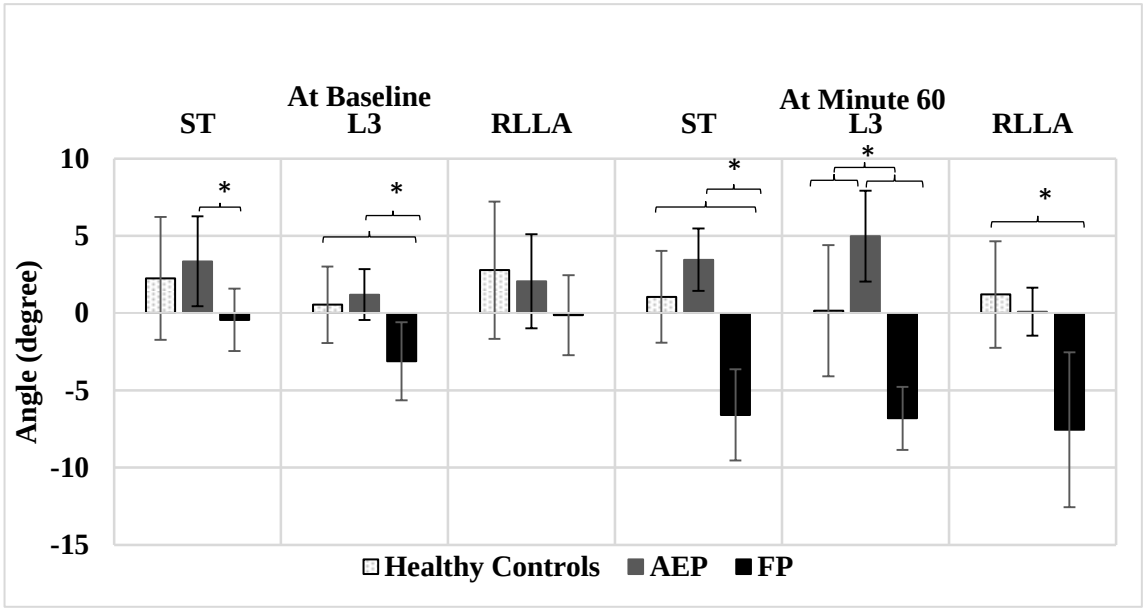



**Figure 1.** Mean (SD) of lumbosacral kinematics at baseline and minute 60 per group (N = 30).

**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; ST, Sacral Tilt; L3, Lumbar 3 Spinous Process; RLLA, Relative Lower Lumbar Angle

(+) angle indicates an anterior sacral tilt/ extension/ lordosis; (-) angle indicates a posterior sacral tilt/ flexion/ kyphosis

*Significant difference ($p \leq 0.05$)

In contrast, all lumbosacral angles differed significantly among the three study groups at the highest level of pain, which was at minute 60 of sitting period ($p<0.001$, $\eta2= 0.74$ for ST, $p<0.001$, $\eta2= 0.72$ for L3 angle, and $p=0.013$, $\eta2= 0.36$ for RLLA). Bonferroni post hoc comparisons revealed that the difference in ST was only significant between FP subgroup and healthy controls ($p<0.001$), and between FP and AEP subgroups ($p<0.001$). Namely, FP had greater posterior sacral tilt (-6.59°±2.95°) compared to healthy controls (1.04°±2.97°) and AEP subgroup (3.45°±2.02°) who exhibited anterior sacral tilt. Furthermore, the difference in L3 angle was significant among all study groups ($p<0.01$). The FP subgroup had greater L3 flexion compared to healthy controls (-6.82°±2.05° vs. 0.15°±4.24°, $p<0.001$) and AEP subgroup who demonstrated L3 extension (-6.82°±2.05° vs. 4.97°±2.94°, $p<0.001$). Also, the AEP had greater L3 extension compared to healthy controls (4.98°±2.94° vs. 0.15°±4.24°, $p=0.007$). Moreover, the difference in RLLA was only significant between FP subgroup and healthy controls ($p=0.020$), and between FP and AEP subgroups ($p=0.048$). The FP subgroup had greater lumbar kyphosis (-7.55°±10.0°) compared to healthy controls (1.20°±5.45°) and AEP subgroup (0.09°±1.56°) who assumed slightly lumbar lordosis.

### *Secondary Analysis*

**Figure 2.** shows the mean of lumbosacral kinematics of all study groups over the 1-hour sitting. There was a significant difference in mean lumbosacral angles (ST and L3 angle) among the three study groups over the entire 1-hour sitting ($p<0.001$, $\eta2= 0.43$, $p<0.001$, $\eta2= 0.70$, respectively). Bonferroni post hoc comparisons showed that the difference in mean ST angle was only significant between the FP and AEP subgroups

($p<0.001$). Specifically, the FP subgroup displayed, at large, a posterior sacral tilt presentation (-1.65°±1.16°), whereas the AEP subgroup exhibited an increased anterior sacral tilt (3.90°±2.70°). In addition, the difference in mean L3 was significant among all study groups ($p<0.01$). The FP subgroup had greater L3 flexion compared to healthy controls (-5.40°±1.60° vs. -0.47°±3.31°, $p<0.001$) and AEP subgroup who demonstrated L3 extension (-5.40°±1.60° vs. 2.92°±1.70°, $p<0.001$). In addition, the AEP had greater L3 extension compared to healthy controls (2.92°±1.70°, vs. -0.47°±3.31°, °, $p=0.009$). However, there was no significant difference in mean RLLA among all study groups ($p=0.412$).

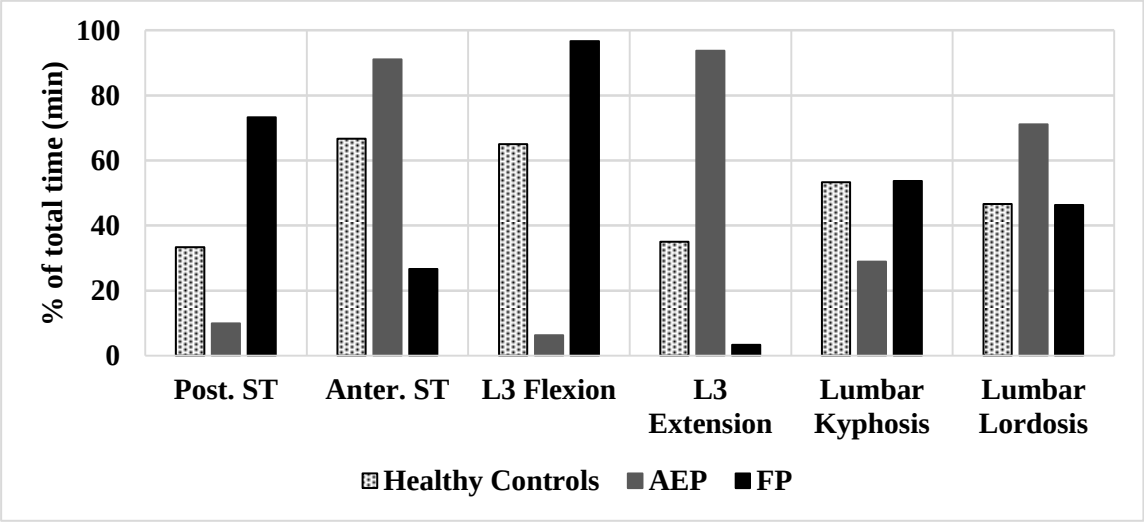



**Figure 2.** Percent (%) of the time spent in the lumbosacral angles per groups (N = 30). Abbreviation: Post., posterior; Anter., anterior; L3, position of the third lumbar spine vertebrae

The results of the analysis of time (min), expressed as a % of the total 1-hour of sitting period, spent in the available ranges of the studied lumbosacral angles showed that the FP subgroup spent more time sitting with posteriorly tilted pelvis (73.3%), whereas the AEP subgroup spent almost all of their sitting time in anterior pelvic tilt (91.1%). In contrast to the pain subgroups, the healthy controls spent 66.7% of their sitting time in anterior pelvic tilt compared to only 33.4% in the posterior direction. In addition, the FP subgroup sat with flexed L3 for 96.7% of the entire sitting period, in contrast, AEP subgroup spent 93.7% of the total sitting time in L3 extension. In comparison to pain subgroups, the healthy controls spent 65.1% of their sitting time in L3 flexion compared to 34.9% in extension. Furthermore, the FP subgroup spent, on average, 53.7% of their sitting time in lower lumbar kyphosis, while the AEP subgroup spent 71.1% of the sitting time in lordosis. Similar to the FP subgroup, healthy controls postured themselves in kyphosis for 53.3% of the sitting time compared to 46.7% in lordosis for the FP subgroup. Refer to **Figure 3.**

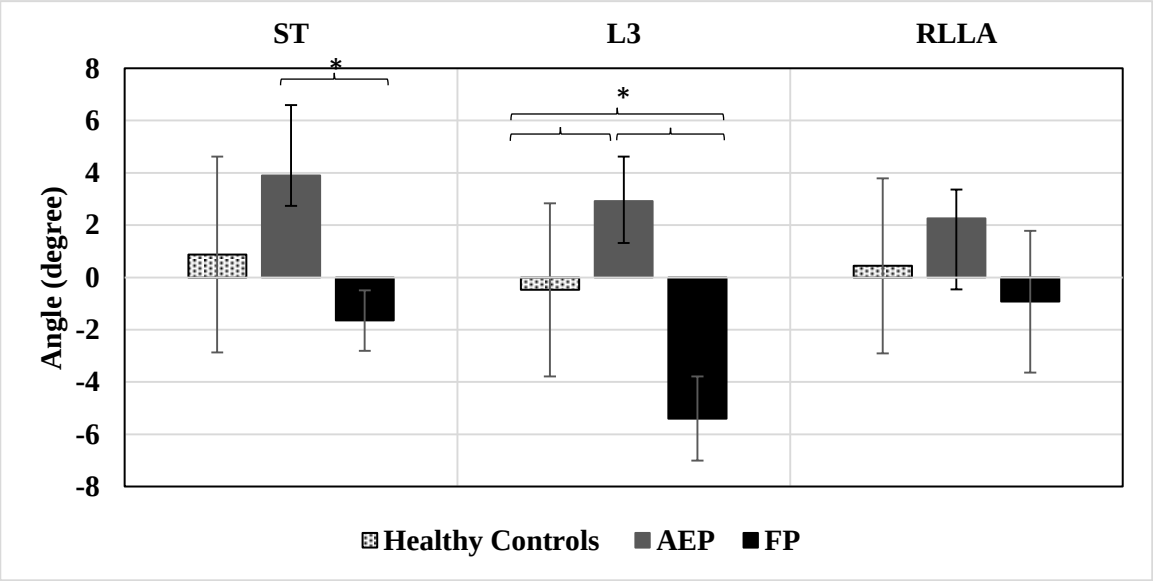



**Figure 3.** Mean (SD) of lumbosacral kinematics over the 1-hour of sitting per group (N = 30).

**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; ST, Sacral Tilt; L3, Lumbar 3 Spinous Process; RLLA, Relative Lower Lumbar Angle

(+) angle indicates an anterior sacral tilt/ extension/ lordosis; (-) angle indicates a posterior sacral tilt/ flexion/ kyphosis

*Significant difference ($p \leq 0.05$).

### *Third Analysis*

**Table 3.** shows the average lumbosacral kinematics and pain by study group overtime. **Figure 4.** shows the average pain scores of all groups over the 1-hour sitting. The mixed factorial analysis showed a significant group by time interaction effect for pain ($p<0.001$, $\eta^2=0.47$). Results of the one-way ANOVA indicated that the difference in the amount of change from baseline was significant among the three groups at all time periods ($p<0.001$). Specifically, Bonferroni post hoc comparisons revealed that both pain subgroups significantly differed from healthy controls at all time periods ($p<0.05$). However, during the first 30 minutes of sitting, pain subgroups did not differ from each other, whereby, during the last 30 minutes, both FP and AEP subgroups were significantly different ($p<0.01$). Namely, the FP subgroup reported a significant increase in pain scores compared to the AEP subgroup at minute 40 (38.10±15.03 vs. 20.00±9.53, $p=0.002$), minute 50 (45.50±18.29 vs. 24.50±16.09, $p=0.007$), and minute 60 (49.20±16.82 vs. 27.40±19.67, $p=0.009$). Similar results were found when adding pain at baseline as a covariate. The level of pain reported by the pain subgroups increased significantly over time ($p<0.001$, $\eta2=0.80$ for FP and $\eta2=0.44$ for AEP), whereby the pain peaked towards the end of the sitting period and increased significantly from baseline after 20 minutes of the sitting period (FP, $p<0.01$ and AEP, $p<0.05$).

**Table 3.** Mean (SD) of lumbosacral kinematics and pain by study group overtime (N=30).

| | FP (n=10) | | | | AEP (n=10) | | | | Healthy Controls (n=10) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Time** | **ST** | **L3** | **RLLA** | **NPRS** | **ST** | **L3** | **RLLA** | **NPRS** | **ST** | **L3** | **RLLA** | **NPRS** |
| **0** | -0.4 (2.0) | -3.1 (2.5) | -0.1 (2.6) | 17.8 (10.1) | 3.3 (2.9) | 1.2 (1.6) | 2.1 (3.0) | 7.9 (6.9) | 2.2 (3.9) | 0.5 (2.5) | 2.8 (4.4) | 0.0 (0.0) |
| **10** | -0.7 (3.1) | -5.7 (1.5) | 0.5 (5.1) | 30.9 (16.7) | 1.9 (5.0) | 2.7 (1.8) | -0.7 (8.7) | 21.1 (16.8) | 0.9 (4.2) | -0.6 (2.6) | 0.3 (5.0) | 0.0 (0.0) |
| **20** | 1.5 (2.8) | -5.3 (2.0) | 4.5 (6.3) | 37.3 (13.6) | 4.3 (3.7) | 2.5 (1.4) | 3.6 (3.6) | 23.6 (13.9) | 0.7 (4.3) | -1.0 (3.5) | -0.3 (6.1) | 0.0 (0.0) |
| **30** | 0.3 (1.2) | -6.5 (2.1) | 1.7 (8.6) | 48.3 (4.7) | 4.6 (2.6) | 3.9 (1.9) | 3.5 (5.9) | 29.8 (18.9) | 0.3 (4.4) | -1.0 (3.7) | -0.7 (6.6) | 0.0 (0.0) |
| **40** | -1.5 (2.8) | -6.2 (1.8) | -0.7 (8.8) | 55.9 (11.2) | 6.0 (2.6) | 1.9 (1.3) | 6.0 (0.9) | 27.9 (12.7) | 0.3 (4.6) | -0.8 (4.5) | -0.5 (7.9) | 0.0 (0.0) |
| **50** | -4.0 (2.9) | -4.2 (5.5) | -4.8 (11.1) | 63.3 (9.8) | 3.8 (2.2) | 3.3 (2.7) | 1.3 (1.4) | 32.4 (2.1) | 0.6 (3.7) | -0.6 (4.0) | 0.3 (7.3) | 0.0 (0.0) |
| **60** | -6.6 (3.0) | -6.8 (2.0) | -7.6 (10.0) | 67.1 (9.0) | 3.5 (2.0) | 5.0 (2.9) | 0.1 (1.6) | 35.3 (23.7) | 1.0 (3.0) | 0.1 (4.2) | 1.2 (5.5) | 0.0 (0.0) |
| **Within group p-value ($\eta^2$)*** | <0.001 (0.57) | 0.017 (0.24) | <0.001 (0.45) | <0.001 (0.80) | <0.001 (0.36) | <0.001 (0.55) | 0.001 (0.33) | <0.001 (0.44) | 0.153 (0.20) | 0.294 (0.12) | 0.057 (0.90) | - |
| **Group x time (p-value, $\eta^2$)**** | ST (0.001, 0.45) | | | L3 (<0.001, 0.34) | | | RLLA (<0.001, 0.36) | | | NPRS (<0.001, 0.47) | | |

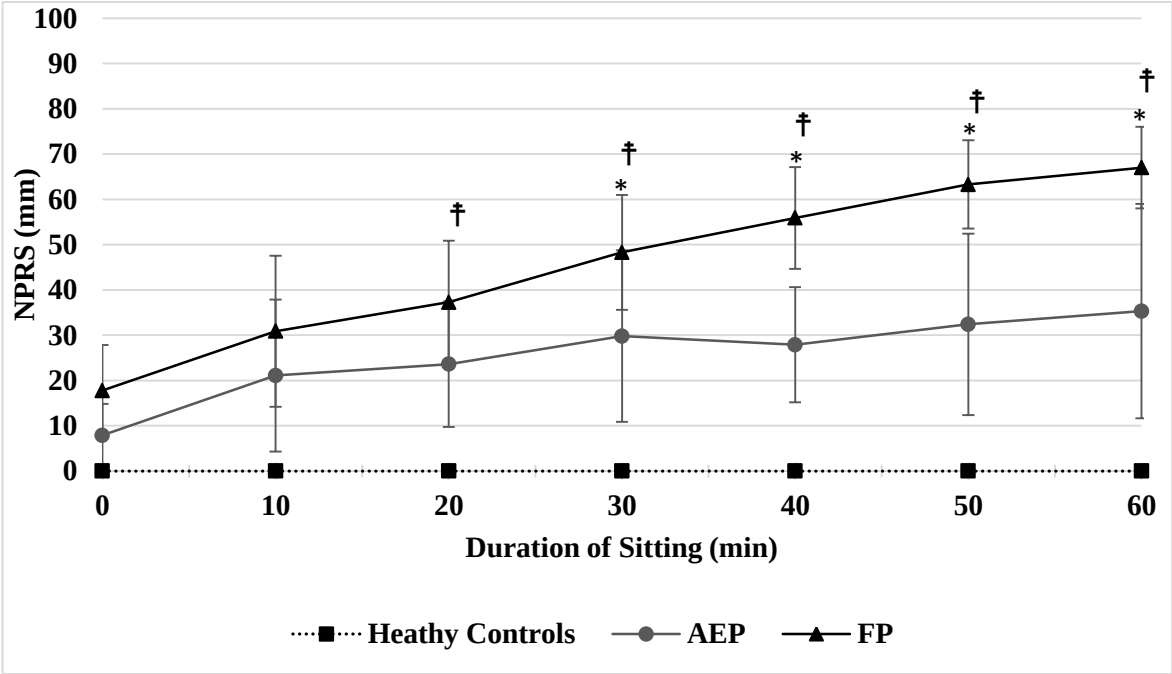
**Abbreviation:** SD, Standard Deviation; FP, Flexion Pattern; AEP, Active Extension Pattern; ST, Sacral Tilt; L3, Lumbar 3 Spinous Process; RLLA, Relative Lower Lumbar Angle; NPRS, Numeric Pain Rating Scale; $\eta^2$, Partial Eta Squared

*One-Way Repeated Measures ANOVA, p≤0.05

**Mixed Factorial ANOVA, p≤0.05

**Figure 4.** NPRS scores over the 1-hour sitting per group (N=30).
**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; NPRS, Numeric Pain Rating Scale

*Significant difference between pain subgroups; †Significant difference from Baseline NPRS ($p \leq 0.05$)



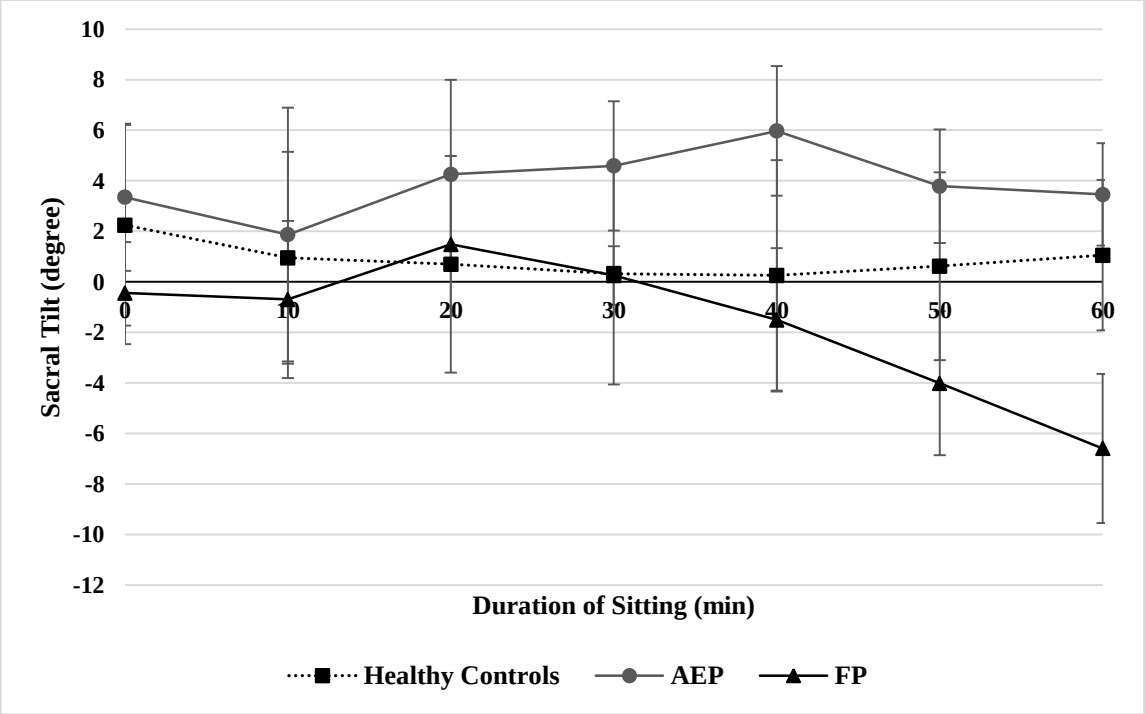
There was a significant group by time interaction effect for ST after controlling for baseline ST angle ($p<0.001$, $\eta 2=0.45$). Results of the one-way ANOVA indicated that the difference in the amount of ST change from baseline was significant among the three groups at minute 20 until the end of the 60-minute sitting period ($p<0.05$). To narrow the results, the amount of ST change from baseline was only reported for the late phase of sitting (at minute 60). Bonferroni post hoc comparisons revealed that the FP subgroup significantly differed from the AEP subgroup ($P<0.001$), and healthy controls ($p=0.001$), but no significant difference was found between AEP subgroup and healthy controls. The degree of ST displayed by the pain subgroups increased significantly over time (FP; $p<0.001$, $\eta 2=0.57$; AEP; $p<0.001$, $\eta 2=0.36$). Specifically, the FP subgroup showed a significant increase in posterior sacral tilt from baseline at only minute 60 (the end of the sitting period) (-0.45°±2.02° vs. -6.59°±2.95°, $p=0.002$), but this change was not statistically significant during the first 50 minutes of sitting. In contrast, the AEP subgroup showed a significant increase in anterior sacral tilt from baseline only at minute 40 (3.35°±2.91° vs. 5.98°±2.03°, $p=0.005$), which then followed by a significant decreased at minute 50 (5.98°±2.03° vs. 3.79°±2.25°, $p<0.001$) and minute 60 (5.98°±2.03° vs. 3.45°±2.03°, $p=0.001$). However, there was no any significant change in ST over time in healthy controls ($p=0.153$, $\eta 2=0.16$). Refer to **Figure 5.a.**

In addition, there was a significant group by time interaction effect for L3 after controlling for baseline L3 angle ($p<0.001$, $\eta 2=0.34$). Results of the one-way ANOVA indicated that the difference in the amount of L3 change from baseline was significant among the three groups at all 6 time periods ($p<0.05$). Bonferroni post hoc comparisons revealed that at minute 60, all groups significantly differed from each other (FP vs.

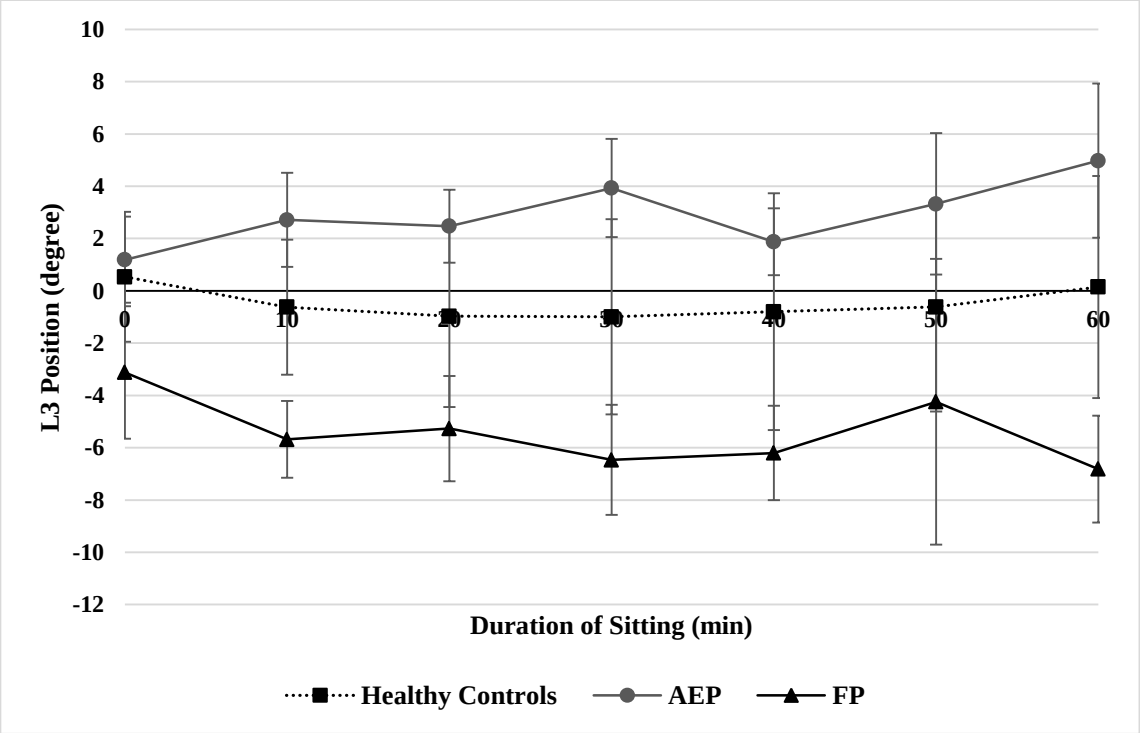
controls, p=0.005; AEP vs. controls, p<0.001; and FP vs. AEP, p<0.001). The degree of L3 displayed by the pain subgroups increased significantly over time (FP; p=0.017, η2=0.24; AEP; p<0.001, η2=0.55). Specifically, the FP subgroup showed a significant increase in L3 flexion from baseline to minute 60 (-3.12°±2.53° vs. -6.81°±2.05°, p<0.001). In contrast, the AEP subgroup showed a significant increase in L3 extension from baseline to minute 60 (1.20°±1.64° vs. 5.01°±2.94°, p=0.001). However, the control group did not show any significant change in L3 over time and remained relatively close to the neutral range (p=0.294, η2=0.12). Refer to **Figure 5.b.**

Furthermore, there was a significant group by time interaction effect for RLLA after controlling for baseline RLLA angle (p<0.001, η2=0.36). Results of the one-way ANOVA showed that the difference in the amount of RLLA change from baseline was significant among the three groups at minute 20 and 40 (p<0.5). Bonferroni post hoc comparisons revealed that at minute 20, the FP subgroup significantly differed from healthy controls (p=0.002), but no significant difference was found between both pain subgroups or between the AEP subgroup and healthy controls. However, at minute 40, only the AEP subgroup significantly differed from healthy controls (p=0.025). The degree of RLLA exhibited by the pain subgroups increased significantly over time (FP; p<0.001, η2=0.45; AEP; p=0.001, η2=0.33). Specifically, the FP subgroup showed a significant increase in lower lumbar kyphosis from minute 20 to minute 60 (4.49°±6.31° vs. -7.55°±10.00°, p=0.038). In contrast, the AEP subgroup showed a significant increase in lower lumbar lordosis from baseline to minute 40 (2.06°±3.04° vs. 5.98°±0.88°, p=0.006), which was followed by a significant decreased at minute 50 (5.98°±0.88°, vs.1.30°±1.41°, p<0.001) and minute 60 (5.98°±0.88°, vs. 0.09°±1.56°, p<0.001).

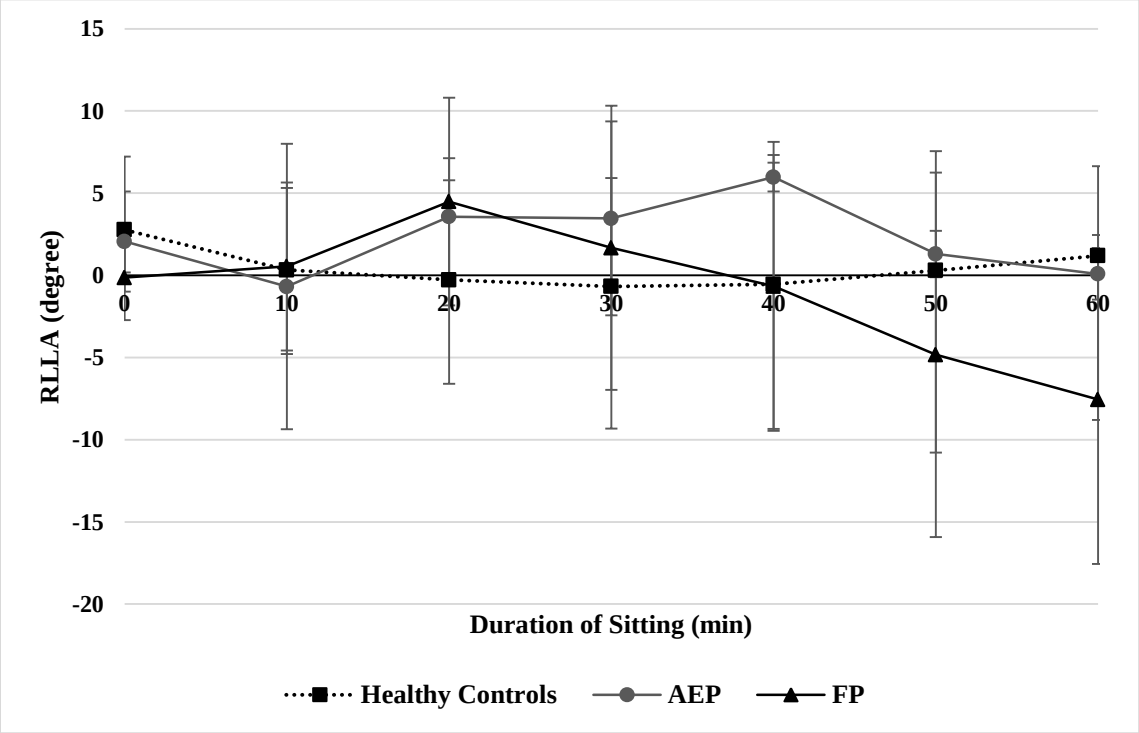
However, healthy controls did not show any significant change in RLLA over time ($p=0.288$, $\eta 2=0.12$). Refer to **Figure 5.c.**




**a.** The amount of change in sacral tilt over the 1-hour sitting (N = 30).




**b.** The amount of change in L3 position over the 1-hour sitting (N = 30).




**c.** The amount of change in Relative Lower Lumbar Angle over the 1-hour sitting (N = 30).

**Figure 5.** The amount of change in lumbosacral kinematics; a. sacral tilt, b. Third Lumbar Vertebrae position, and c. Relative Lower Lumbar Angle over the 1-hour sitting (N = 30).
**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; ST, Sacral Tilt; L3, Lumbar 3 Spinous Process; RLLA, Relative Lower Lumbar Angle
**For the sacral tilt:** (+) angle indicates an anterior sacral tilt; (-) angle indicates a posterior sacral tilt
**For the L3 position:** (+) angle indicates an extension; (-) angle indicates a flexion
**For the RLLA:** (+) angle indicates a lordosis; (-) angle indicates a kyphosis

# Discussion

## *Summary of the findings*

The present study aimed to investigate spine postural behaviors among two commonly studied MCI subgroups (FP and AEP) compared to heathy controls, and their role on pain development during a 1-hour of prolonged sitting. The results of this study showed that all study groups presented with significantly distinctive postural behaviors at the beginning and at the end of the sitting period. Only the MCI subgroups, however, showed significant deterioration in the lumbosacral kinematics and pain levels overtime. The direction of deterioration in lumbosacral kinematics over the 1-hour sitting period occurred in the direction of the motor control impairment (kyphosis for FP or lordosis for the AEP subgroup). Interestingly, both MCI subgroups reported a similarly significant increase in pain through mid-sitting. However, after mid-sitting, the AEP subgroup displayed a significantly reversed decrease in the lordotic posture which was accompanied by much less increase in pain level compared to the FP subgroup. The findings of this study suggest a possible association between the lumbosacral postures and pain development overtime.

## *FP subgroup*

In the present study, the FP subgroup exhibited an increased kyphotic presentation of the lower lumbosacral region over the entire 1-hour of sitting period as compared to the other groups. Interestingly, the observed differences in kinematics did not only appear after the onset of increased pain, instead, they were initially present at baseline, which may further suggest an inherent postural behaviors in the FP subgroup that predisposed

them to pain (15, 33). In addition, these behaviors continued to deteriorate in the direction of flexion throughout testing, which could imply an overtime loss of the motor control of the moving lower lumbosacral segments in the direction of pain provocation (9) contributing to the increased pain overtime. Furthermore, over the 1-hour sitting period, FP subgroup did not show any attempt to produce a positional alteration in order to reduce their pain, instead, they maintained a directional increase of the lower lumbosacral flexion. This might further support the presence of a "neutral spinal position deficit" in which they underestimated the neutral position of lower lumbar by adopting a kyphotic posture (34) throughout the sitting period resulting in pain provocation. Lastly, the FP subgroup spent the majority of their sitting, in general, at end-range flexion posture. Maintaining an end-range posture from the beginning through the end of sitting period, as shown clearly in the present study, was accompanied by a significant increase in pain overtime. Specifically, the pain increased to statistically significant level from the beginning of the sitting period after >20 minutes and remained significant for the rest of the sitting period. This could suggest that the exhibited flexion end-range behavior might have caused a progressive increase of the lower lumbar strain in the FP subgroup, disrupting the physiological distribution of spinal loading in lumbosacral structures, leading to the incremental increase in pain over the sitting period (15, 33).

Similar to these findings, previous studies reported a greater posterior sacral tilt (15, 16) and flexion of the lower lumbar angle (15) in the FP subgroup during a 5-second, 1-hour sitting (17), and field cycling (33). Similar to the findings of the present study, the cited studies attributed pain increase to the sustain extreme flexion posture in the FP subgroup (17, 20, 35) secondary to: a) inherent motor control impairments in the

direction of flexion, b) proprioceptive alterations of spine structures (34), and c) restricted lumbosacral range of movement toward the opposite direction of the motor control deficits (15). Lastly, it is important to note that the present study determined the direction of the postural control impairment over a 1-hour sitting period and identified its influence in pain increase within the FP subgroup. This could further clarify the underlying mechanism of pain development and eventually assist in the development of customized chairs or biofeedback training approaches into clinical practice to address these postural faults adopted by this subgroup.

### *AEP subgroup*

In contrast, the AEP subgroup exhibited an increased lordotic posture of the lower lumbosacral region over the entire 1-hour of sitting period as compared to the FP and healthy controls. Similar to the FP subgroup, the observed differences in kinematics were present at baseline and evidently appeared after the onset of pain, which again may further support the presence of the inherent postural behaviors in the AEP subgroup (15, 33). In the same manner to the FP subgroup, the observed postural behaviors continued to deteriorate in the direction of extension but only throughout the early phase of sitting (<40 minutes), which may suggest an overtime loss of the motor control of the lower lumbosacral segments (9) contributing to the increased pain at the early phases of sitting. However, after 40 minutes of sitting, the AEP subgroup showed some positional alteration toward “neutral” which could be interpreted as an adaptive approach to reduce/control their pain level. This finding might further support that the AEP subgroup possibly had a “neutral spinal position deficit” only at the early phase of sitting period, in

which they underestimated the neutral position of lower lumbar by adopting a lordotic posture (34) resulting in pain provocation. However, less positional deficits were observed after 40 minutes of sitting as the AEP subgroup moved toward “neutral”. Lastly, the AEP subgroup spent most of their early sitting time at end-range extension. Sustaining an end-range postures from the beginning through the mid-point of sitting period was associated with a significant increase in pain. Specifically, the pain increased to statistically significant level from the beginning of the sitting period after >20 minutes through the end. Although, the pain after 40 minutes was significantly different from baseline, it was slightly lower compared to the FP subgroup. The initial lordotic behavior over the 1-hour sitting may contribute to the increased extension compressive force, developed by sustained lumbar extension and possible muscle fatigue, in posterior spinal structures resulting in development of pain (15). However, the later correction of the postural faults toward neutral might explain the noted reduction in pain level toward the end of the sitting period in this MCI subgroup.

Similar to these findings, previous studies reported a greater anterior sacral tilt (15, 16) and extension of the lower lumbar angle (15) in the AEP subgroup during 5-second sitting, 10-minute (20), and functional tasks (11). Although sitting in a standard chair might have promoted lower lumbar flexion in the AEP subgroup (20), ironically they assumed hyperextension postures away from the neutral spectrum resulting in pain increase in the first 40 minutes of sitting. However, toward the end of sitting period they assumed more of a neutral posture which was associated with relatively lesser back pain compared to the FP subgroup. The initial lordotic posture could be explained by reduced ability of the AEP subgroup to relax their paraspinal muscles which in return might have

minimizing their ability to tilt their pelvis posteriorly (13, 20). Unfortunately, the lack of muscle activation data hindered the ability to confirm this notion. The latter "neutral posture" is in line with the previously reported findings by Curran et al. (2014) [20], in which the AEP subgroup reported greater back discomfort when they sat on a forward inclined seatpan, but the pain was lower while sitting on a flat seatpan chair. The findings from this study could imply that the positions of lumbosacral region in the AEP subgroup were maybe related to pain alterations.

### *Study Limitations*

The levels of trunk muscles activation were not measured in the present study, which might have omitted their influence in spine postures, although previous studies found inconsistent differences in muscle activities among the MCI subgroups (34) or when they are compared to healthy controls (20). In addition, the sitting period was only monitored for an hour, which might not have provided a thorough understanding of how MCI subgroups operates during their daily office tasks that extends beyond an hour. However, due to a) the likelihood of experiencing LBP by these subgroups over a single hour, and b) the logistic of the testing, it was intended to limit the sitting period to an hour to avoid unacceptable pain aggravation. Furthermore, the association between pain levels and lumbosacral kinematics were not analyzed, however, the purpose of this study was to establish the differences among the MCI subgroups in lumbosacral kinematics prior to the onset of pain increase and at the highest levels of pain over an hour of sitting, and thus the correlation analysis will be performed in depth in a future publication to understand the nature of this relationship. Also, sitting posture might not be challenging

enough for the AEP subgroup to produce pain due to its flexed nature (15-17), however, in the present study the AEP subgroup reported an increase in pain level over the 1-hour sitting. This can be attributed to the static loading at the lumbosacral spine associated with the prolonged sitting period making it a provocative means to aggravate pain in both studied MCI subgroups. Although the FP and AEP disorders are sagittal plane motor control deficits, so the two-dimensional inclinometer would sufficiently capture the deviations from the sagittal plane, future studies are warranted to monitor regional and segmental spine postures in the three planes of movements overtime. Unfortunately, this was not available for the present study.

### *Clinical Implications*

The findings of the present study highlight the postural behaviors that NSCLBP patients with MCI display while sitting for an extended period of time. Identifying these behaviors and their contributions to pain development might refine the application of the classification-based cognitive functional therapy (CB-CFT) (14) in FP and AEP subgroups. A postural biofeedback training to facilitate proper lumbosacral kinematics away from the end-range sitting postures, could be relevant in spine rehabilitation for these subgroups (14, 36). Also, incorporation the findings to intervention approaches for these subgroups might advance NSCLBP management. For example, ergonomic recommendations regarding the use of a lumbar roll for the FP subgroup and a declined seatpan for the AEP subgroup might assist in pain reduction among the MCI subgroups.

## *Conclusion*

The results of this study showed that both MCI subgroups presented with distinctive underlying maladaptive postural patterns. However, the significant increase in pain over the 1-hour sitting might not be only attributed to the inherent maladaptive postures, also it may be related to the directional deterioration in lumbosacral postures overtime. Incorporating these findings into treatment strategies might assist in reducing sitting back pain among MCI subgroups.

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# CHAPTER THREE

# NON-SPECIFIC CHRONIC LOW BACK PAIN: EXAMINING TRUNK MUSCLE ACTIVITY DURING PROLONGED SITTING IN SUBGROUPS OF MOTOR CONTROL IMPAIRMENT; A CROSS-SECTIONAL STUDY


## Abstract

### *Background*

Although, non-specific chronic low back pain (NSCLBP) has been related to abnormal trunk muscle activations, little is known about the possible driving mechanisms of pain development overtime during prolonged sitting period. Therefore, the purpose of this study was to examine the differences in muscle activity in adults with and without NSCLBP, and their role on pain development during a 1-hour of prolonged sitting task.

### *Methods*

Twenty NSCLBP subjects with motor control impairment (MCI) [10 classified as having flexion pattern (FP) disorder, and 10 with active extension pattern (AEP) disorder], and 10 healthy controls participated in the study. Subjects followed a 1-hour sitting protocol on a standard office chair. Four trunk muscle activities including amplitudes and co-contractions were recorded using electromyography over the 1-hour period. Perceived back pain intensity was recorded using a numeric pain rating scale every 10 minutes throughout the sitting period.

### *Results*

All study groups presented with no significantly distinctive trunk muscles' activities at the beginning of sitting ($p>0.05$), nor did they change overtime when pain increased to a significant level ($p>0.05$). Both MCI subgroups reported a similarly

significant increase in pain through mid-sitting ($p<0.001$). However, after mid-sitting, they significantly differed from each other ($p<0.01$).

***Conclusion***

The study's findings suggest that people with and without NSCLBP related to MCI presented with similar muscular patterns, and the significant increase in pain over the 1-hour sitting might not be attributed to trunk muscles' activation.

***Keywords***

Low back pain, Motor control impairment, Flexion pattern disorder, Active extension pattern disorder, Prolonged sitting, Muscle Activity.

## Introduction

Low back pain (LBP) is a common health disorder related to physical, social and economic burden, and it has been recognized as the leading cause of disability [1, 2]. Its prevalence per month is approximately estimated to be 23% in the general population and continues to rise [3]. Although, most LBP cases recover within 3 to 4 weeks [4-6], a large percentage continues to report long lasting disability related to chronic pain [6-8]. Approximately 85% of these chronic LBP (CLBP) conditions are often known as non-specific CLBP (NSCLBP) due to the inability to identify a specific pathology [6, 7].

Abnormal neuromuscular control and its contribution to pain development has been well documented in patients with NSCLBP [9, 10]. Despite the considerable amount of evidence suggesting the presence of motor control faults in NSCLBP patients, the nature of these faulty patterns in response to pain provocation are highly inconsistent [11-15]. Several research studies reported no differences in trunk muscle activations in this population [11, 14, 15]. In contrast, one study reported a decrease in muscle activations

in patients with NSCLBP [16] while others reported an increase in muscle activities [10, 17]. These inconsistent findings were commonly attributed to the "washout effect" when interpreting EMG data of heterogeneous CLBP patients [12]. Therefore, a Multidimensional Classification system (MDCS) emerged in which, a large number of those patients was classified based on the underlying mechanical basis of their pain disorder [9, 11, 12, 18-20].

A study that used the MDCS, found that the levels of activity of lumbar muscles were higher in extension-related NSCLBP, namely active extension pattern (AEP) subgroup, when compared to healthy subjects and flexion-related back pain, called flexion pattern (FP) subgroup during 5-second sitting [12]. This increase in back muscle activity is thought to predispose those patients to pain [9, 12]. Contrary to that, muscular patterns of NSCLBP patients were found highly variable in other research studies using the MDCS [11, 14, 15]. Most of these studies examined the neuromuscular functions of the trunk muscles using activation amplitudes as an indicator for the motor response [11-15]. However, a muscle pairing analysis (an analysis of localized co-contraction) was not considered. This type of analysis allows a better understanding of the activation pattern of one abdominal and another back muscle at the time of a specific activation, which allows to thoroughly understand the relationships between these two paired muscles during known tasks [17, 21].

So far, research data showed inconsistent differences in trunk muscle amplitudes among MCI subgroups; however, co-activation analysis has not been previously performed for the MCI subgroups. Therefore, identifying co-contraction patterns and understanding the relationship between abdominal and back muscles among the MCI

subgroups in presence of pain provocative would enable clinicians to provide customized, subgroup-specific interventions based on the underlying motor control impairments. Furthermore, patients with MCI, FP and AEP subgroups, demonstrate pain provocation during the exposure to static loading such that occurring during prolonged sitting [9]. Because prolonged sitting is frequently reported to aggravate pain in NSCLBP population [17, 18], research examining sitting nature is becoming increasingly relevant. Therefore, the purpose of this study was to examine whether the trunk muscles' co-contraction a) differed among FP, AEP subgroups and healthy controls, and b) changed overtime when pain is at its lowest and at its highest over a 1-hour sitting task. It was hypothesized that subjects with FP and AEP disorders would display higher coactivation patterns as the pain is at its lowest level when compared to healthy controls, and that these patterns would increase toward the end of sitting period as the pain is at its highest level.

## Materials and Methods

### *Participants*

A total of 30 subjects; 20 NSCLBP with MCI and 10 healthy were recruited from private outpatient Physical Therapy clinics. Ethical approval was obtained from the Institutional Review Board at Loma Linda University (LLU) #5180306. All subjects read and signed a written informed consent prior to participation in the study. To establish MCI sub-classifications, two therapists independently completed a comprehensive subjective assessment and physical examination [18]. Only subjects with FP or AEP

based on the criteria explained elsewhere were included in the study [9, 22]. Inclusion and exclusion criteria are summarized in **Table 1.**

**Table 1.** Inclusion and Exclusion Criteria

| Inclusion Criteria | Exclusion Criteria |
|---|---|
| • ≥ 3 months NSCLBP<br>• ≥ 5 points scored on RMDQ score<br>• Pain in the lower lumbosacral region<br>• Absence of "red flags" (such as inflammatory diseases or causa equina)<br>• Absence of dominant "yellow flags" (such as identification of beliefs, emotions, and behaviors that interact with the pain problem)<br>• Clear mechanical basis of disorder<br>• Associated impairments in the control of the motion segment(s) in the provocative movement direction (s)<br>• Absence of impaired movement of the symptomatic segment in the painful direction of movement (based on clinical joint mobility examination)<br>• Diagnosis of an FP or AEP disorder (both examining clinicians independently agreed upon the diagnosis) | • <5 points scored on RMDQ score<br>• Signs of neurologic involvement, e.g., radicular pain, and more generalized pain<br>• Evidence of specific diagnosis, e.g., spondylolisthesis, inflammatory disease,<br>• Previous spine surgery<br>• Pregnant at the time of the study or 6 months postpartum |

**Abbreviation:** NSCLBP, non-specific chronic low back pain; RMDQ, Ronald Morris Disability Questionnaire; LBP, low back pain; FP, flexion pattern; AEP, active extension pattern.

Gender, age, anthropometric data [mass, height, and body mass index (BMI)], perceived pain using the visual analogue scale (NPRS) [17, 23, 24], pain duration, , pain-related disability using Ronald Morris Disability Questionnaire (RMDQ) [25] were collected at baseline**.** Data collection was conducted at the Orthopedic and Movement Science Laboratory, Department of Physical Therapy, LLU, California, United States.

## *Instrumentation*

### Electromyography (EMG)

An 8-channel MyoMuscle 1200 EMG system (Noraxon USA, Inc, Scottsdale, AZ) with an input impedance of greater than 100 mΩ, a gain of 500, and a common-mode rejection ratio of greater than 100 dB was used to record muscle activity during a 1-hour siting protocol. EMG signals were acquired at a sampling rate of 1000 Hz. In accordance with previous literature, EMG activity of the local lumbosacral stabilizers (EO, TrIO, sLM [12] and LES [17]) was recorded.

### Perceived Pain

While it is challenging to measure pain [24], perceived pain scores was gathered using the open numeric pain rating scale (NPRS) during the 1-hour sitting protocol . The NPRS consists of a 100 mm horizontal line, anchored by the descriptors “no pain” and “worst pain imaginable” [23].

## *Procedures*

### Placement of EMG Electrode

Prior to electrode placement, subjects' skin was shaved, abraded, and cleaned with isopropyl alcohol wipes. Disposal surface electrodes (dual, 2 mm diameter, 2 cm apart, Noraxon USA, Inc) were placed parallel to the muscle fibers in accordance with the SENIAM research group recommendations and previous research [12, 26, 27]. The external oblique (EO) electrode was placed below the rib cage and along a line connecting the opposite pubic tubercle and the most inferior point of the costal margin [28]. The transfer fibers of internal oblique (TrIO) electrode was placed 1 cm medial to the anterior superior iliac spine (ASIS) and beneath a line connecting both ASISs [17, 28]. The lumbar erector spinals (LES) electrode was placed 4 cm lateral to the spinous process of L3 [27]. The superficial lumbar multifidus (sLM) electrode was placed at the level of L5 along a line joining posterior superior iliac spine (PSIS) and L1–L2 interspinous space [17]. The same therapist placed all electrodes to ensure consistency. EMG sensors were positioned using a double-sided tape and further secured to the skin with an adhesive tape to minimize movement artifacts during the testing. Electrodes' placement was visually confirmed by viewing EMG signals during a manual muscle test to minimize crosstalk effect.

### Sub-Maximal Voluntary Contraction (sub-MVC) Evaluation

For the EO and TrIO testing, subjects were positioned in crook lying with both hips flexed to 45° and the knees flexed to 90° [29]. To record sub-MVC, subjects were then asked to raise both legs 1 cm off the supporting table for 3 seconds. For the LES and

sLM testing, subjects were positioned in the prone lying position with both knees flexed to 90°. To record sub-MVC, subjects were asked to lift both thighs 5 cm off the table for 3 seconds [29]. Subjects were instructed to avoid quick contraction and to gradually build up their effort to their maximum once they hear the word ªGo!". Prior to the measurement trials, subjects completed 1 practice sub-MVC trial to ensure adequate performance. Three 3-second measurement trials were performed for each muscle with a 3-minute rest period in between each trial to minimize fatigue or aggravation of back pain. All subjects received a standard verbal encouragement during each trial. An additional trial was taken if an arbitrary value of more than 10% of variation between the three trails was noticed to avoid large variability. The same therapist completed all measurements to ensure consistency, and the order of muscle testing was randomized to minimize bias.

**Pain Measurement**

Perceived pain was collected immediately prior to the beginning of the sitting protocol, and every 10 minutes throughout the 1-hour. This way, we obtained a total of seven readings for each subject. All subjects were asked to rate their pain by making a vertical line in the open NPRS at the point corresponding to their level of pain/discomfort. To avoid artificial increase in the NPRS scores, all subjects were asked to focus on pain intensity rather than the locations of their pain [17, 24]. Additionally, subjects were allowed to compare their current NPRS score with the preceding scores to minimize unintended rating variations when drawing lines corresponding to their pain [24].

**1-Hour Sitting Protocol**

Following sub-MVIC evaluation, all subjects underwent a 1-hour sitting protocol in which they sat on an office chair reading pre-selected passages. Prior to sitting, the chair was modified by removing the backrests and armrests so they do not interfere with data collection [17]. Although, this might alter the sitting behavior of the subjects, research found no difference in the trunk muscle activity when sitting in an office chair with backrest or on a stability ball without backrest [30]. Therefore, this modification is deemed to be appropriate. In addition, the height of the chair was adjusted so that the subjects sat with hips and knees approximately at $90^0$ of flexion [17] and feet rested on the floor [31]. Subjects were then provided with a standard office workstation setup, including a monitor, with the top of the screen at eye level, a keyboard and a mouse [17]. Subjects were instructed to follow the text on the monitor screen with the mouse cursor using the right hand and pressing the 'Shift' key on the keyboard to move to the next paragraph using the left hand [17, 32]. The subjects' elbows were kept at $90–100^o$ of flexion while reading, thus the height of the keyboard and mouse was adjusted to maintain this elbow angle [31]. The distance from the keyboard was standardized for all subjects, in which the edge of the keyboard was in line with the radial styloid process and a distance of approximately 30 cm to subjects' greater trochanter [31]. Thus, the potential for confounding variables affecting the study findings were minimized. Finally, just prior launching the 1-hour sitting protocol, all subjects were instructed to "sit as they normally will' on their office chairs.

Over the 1-hour sitting protocol, the amplitude value for each muscle activity [17, 24] was recorded, and pain level [17, 24] were recorded at baseline and every 10 minutes throughout the sitting protocol.

## *Data Processing*

### EMG Activation Amplitudes

Before processing raw EMG data, visual inspection was utilized to eliminate potential artifacts. Then all data were processed on Noraxon EMG system in which the signals were high-pass filtered using a dual-pass Butterworth filter with a cutoff frequency of 30 Hz, in order to remove contamination by heart rate and other artifacts [33]. Signals were then full-wave rectified, low-pass filtered using a dual-pass Butterworth filter with a cutoff frequency of 2.5 Hz [12, 17, 34]. To establish the MVIC, the highest peak value out of the three trials for each muscle from the sub-MVIC evaluation was automatically selected as sub-MVIC and used for normalization. During normalization, the average amplitude values of each muscle over the entire 1-hour sitting period and at every 10-minute interval were normalized to the sub-MVIC, expressed as a percentage (%), and used for the analysis.

### Reliability of the Measurements

The standard error of measurement was used to assess the reliability of measurement [10, 12]. The intertrial reliability of the obtained EMG data was good. The standard error of measurement ranged from 0.09 to 0.24 (% of sub-MVIC).

### EMG Co-contraction Index (CCI)

A co-contraction index was used to measure the level of activations and timing of these activations of two trunk muscles (abdominal_back pairing) [12]. The processed

EMG data, as described above, was then used to calculate the co-contraction indices (CCIs) over the entire 1-hour sitting period and every 10-minute interval. Since CCI reflects the activation level of two paired muscles, muscle pairings were needed. To establish muscle pairings, each abdominal muscle was paired with each back muscle resulting in 16 possible pairings per subject at each time period. The muscle pairs reflect abdominal-back coactivations [17], and are as follows (REO_RLES, REO_LLES, REO_RLMS, REO_LLMS, LEO_RLES, LEO_LLES, LEO_RLMS, LEO_LLMS, RIO_RLES, RIO_LLES, RIO_RLMS, RIO_LLMS, LIO_RLES, LIO_LLES, LIO_RLMS, LIO_LLMS). The CCI values of each pairing were calculated using the equation [1] revealing a total of 128 values (8 CCI's values per pairing). Microsoft Excel (Version 16.25) was used to perform the calculation.

$$\mathrm{CCI} = \sum_{i-1}^{n} \left(\frac{EMG_{low(i)}}{EMG_{high(i)}}\right) \mathrm{x} \left[\mathrm{EMG_{low(i)}} + \mathrm{EMG_{high(i)}}\right] \qquad [1]$$

**Numeric Pain Rating Scale (NPRS)**

Post-collection, the perceived pain (a total of 7 readings) was used for analysis. For the pain subgroups, the highest NPRS score was used to determine which data to be used for the primary analysis for each subject. For example, if a subject reported the highest pain level at the third 10-minute interval, then only CCIs of the trunk muscles at the beginning of the sitting period (baseline) and third 10-minute interval were used for comparison within the same group and among study groups. This way, we were able to compare the trunk muscles activation of each subject when the pain was at its lowest and highest pain levels.

## Statistical analysis

Data was summarized using mean and standard deviation (SD) for quantitative variables and counts (%) for qualitative variables. The normality of continuous variables was examined using Shapiro Wilk's test and Q-Q normality plots. The distribution of the subjects' characteristics by study group were evaluated using chi-square test for independence for gender, one-way Analysis of Variance (ANOVA) for age, height, mass and BMI, and independent t-test for muscle amplitudes between sides (right vs. left), duration of pain, and Kruskal Wallis ANOVA for NPRS (during past 24 hours, past week, and baseline), TSK and RMDI scores.

The primary analysis included a comparison of trunk muscles' amplitudes and CCIs of each pair across groups at the lowest (baseline) and highest level of pain (minute 60) using one-way ANOVA (with post-hoc Bonferroni). The secondary analysis included a comparison of muscles' amplitudes and CCIs of each pair across groups over the entire 1-hour sitting using one-way ANOVA (with post-hoc Bonferroni). A third analysis included a 3x7 mixed factorial ANOVA (between factor: group; within factor: time) to examine changes in trunk muscles amplitudes and CCIs of each pair, and NPRS by study group over time. If the group x time interaction effect in the mixed factorial ANOVA was statistically significant, change from baseline was compared among groups at each time period (total of six "10-minute intervals") using one-way ANOVA (with post-hoc Bonferroni). If the interaction was not statistically significant, the between-groups comparison was considered not statistically significant. However, if the main effect of time was significant in the mixed factorial ANOVA, a one-way repeated measures ANOVA (with post-hoc Bonferroni) was used to examine changes over time within-

groups separately. The level of significance was set at $p \leq 0.05$. Statistical analysis was performed using IBM SPSS Software version 25 for Windows (Chicago, IL, USA).

### *Sample size estimate*

For the primary and secondary analyses, a sample size of 30 subjects was estimated using a large effect size [eta squared ($\eta^2$) = 0.26], level of significance ($\alpha$ = 0.05), and power of 0.80. For the third analysis, a sample size of 30 subjects was estimated using a moderate effect size for the group x time interaction (partial $\eta^2 = 0.06$), level of significance ($\alpha = 0.05$), and power of 0.90.

## Results

A sample of 30 subjects with mean age 27.6±4.9 years, mass 151.9±30.3 lbs., height 5.4±0.4 feet, BMI 25.0 ±3.9 kg/m$^2$ participated in this study. Fifty-seven percent of the subjects were males (n=17). The distribution of all quantitative variables was approximately normal. There was no significant difference in subjects' characteristics by study group ($p>0.05$). Subjects' characteristics are summarized in **Table 2.** Additionally, independent t-test revealed no differences between right and left side muscle amplitudes, thus, the muscle amplitude results were reported from one randomly selected side (right**).**

**Table 2.** Mean (SD) of Baseline Characteristics by Study Group (N = 30).

| | **FP (n-10)** | **AEP (n=10)** | **CG (n=10)** | **p-value** |
|---|---|---|---|---|
| **Male (n)** | 7 | 4 | 6 | 0.39 |
| **Age, y** | 27.8 (4.0) | 27.9 (5.3) | 27 (5.8) | 0.91 |
| **Height, feet** | 5.8 (0.3) | 5.3 (0.3) | 5.2 (0.5) | 0.06 |
| **Mass, lbs** | 157.5 (30.3) | 154.4 (36.1) | 143.8 (25.0) | 0.58 |
| **BMI ($kg/m^2$)** | 24.8 (4.5) | 25.0 (4.4) | 25.2 (2.9) | 0.98 |
| **NPRS (average/week/100mm)** | 45.3 (14.1) | 40.0 (19.2) | - | 0.50 |
| **NPRS (average/24hr/100mm)** | 19.7 (13.8) | 12.0 (10.9) | - | 0.31 |
| **NPRS (average/Baseline)** | 17.8 (10.1) | 7.9 (6.9) | - | 0.02 |
| **Pain Duration, year** | 3.5 (5.3) | 6.0 (5.6) | - | 0.33 |
| **RMDI (%)** | 7.2 (2.2) | 6.0 (1.5) | - | 0.24 |
| **TSK (64 score)** | 14.2 (5.7) | 22.8 (8.4) | - | 0.06 |

**Abbreviation:** SD, Standard Deviation; FP, Flexion Pattern; AEP, Active Extension Pattern; CG, Control Group; BMI, Body Mass Index; NPRS, Numeric Pain Rating Scale; RMDI, Ronald Morris Disability Index; TSK, Tampa Scale of Kinesophobia

### *Primary Analysis*

The differences in muscle activation amplitudes among study groups at the beginning and the end of 1-hour sitting are displayed in Table 3. There was a significant difference in mean REO activation amplitude among the three study groups at the lowest level of pain, which was at beginning of the sitting period ($p=0.028$, $\eta^2= 0.24$). Bonferroni post hoc comparisons showed that the difference in mean REO was only significant between the AEP subgroup and healthy controls ($p=0.026$). The AEP subgroup had lower mean REO activation compared to healthy controls (REO: 0.36±0.10 vs. 0.69±0.35). However, there was no significant difference in mean REO activations between FP and healthy controls ($p=0.23$).

Also, a significant difference was only noted in mean REO activation amplitude among the three study groups at the highest level of pain, which was at minute 60 of the sitting period ($p=0.050$, $\eta^2= 0.20$). Bonferroni post hoc comparisons revealed that the difference in mean REO activation was only significant between AEP subgroup and healthy controls ($p=0.046$). Specifically, AEP had lower activation compared to healthy control (0.38±0.19 vs. 0.80±0.50, $p=0.046$). However, there was no significant difference in mean REO activation between FP and healthy controls ($p=0.79$).

No significant differences in mean activation amplitudes were found for the other muscle groups ($p>0.05$) as well as, no significate differences were noted for CCIs at baseline or at minute 60 ($p<0.05$).

**Table 3. a.** Mean (SD) of trunk muscles' amplitudes and cocontraction indices (expressed as %sub-MVC) at baseline per group (N=30)

| Muscle | Baseline | | | p-value ($\eta^2$) |
|---|---|---|---|---|
| | Healthy Controls | AEP | FP | |
| **RIO** | 0.30 (0.32) | 0.40 (0.28) | 0.51 (0.35) | 0.350 (0.21) |
| **REO** | 0.69 (0.35) | 0.36 (0.10) | 0.53 (0.27) | 0.028 (0.24)* |
| **RLMS** | 0.20 (0.10) | 0.20 (0.07) | 0.22 (0.14) | 0.807 (0.19) |
| **RLES** | 0.22 (0.12) | 0.21 (0.13) | 0.32 (0.13) | 0.132 (.012) |
| **REO_RLES** | 8195.45 (4547.08) | 7866.84 (4753.40) | 12554.45 (8031.90) | 0.218 (0.23) |
| **REO_LLES** | 6407.00 (3606.06) | 7242.14 (3479.50) | 7541.62 (2327.13) | 0.741 (0.02) |
| **REO_RLMS** | 7847.93 (4255.26) | 7248.30 (2594.70) | 9118.73 (6131.55) | 0.704 (0.21) |
| **REO_LLMS** | 5024.40 (2277.76) | 6723.51 (3253.10) | 6855.32 (3502.94) | 0.359 (0.19) |
| **LEO_RLES** | 7254.40 (3008.30) | 7014.93 (3074.53) | 12057.00 (6742.03) | 0.055 (0.04) |
| **LEO_LLES** | 6329.62 (3526.00) | 6799.54 (2602.23) | 7261.15 (2981.01) | 0.819 (0.23) |
| **LEO_RLMS** | 7256.80 (3589.05) | 6811.62 (2540.51) | 8666.15 (5668.21) | 0.642 (0.20) |
| **LEO_LLMS** | 5059.75 (2077.35) | 6078.92 (2335.83) | 6765.51 (4274.60) | 0.485(0.19) |
| **RIO_RLES** | 5452.63 (3770.57) | 7268.12 (4744.20) | 10839.31 (8110.73) | 0.156 (0.22) |
| **RIO_LLES** | 5596.31 (4638.30) | 6501.70 (3253.22) | 7148.03 (2318.21) | 0.667 (0.25) |
| **RIO_RLMS** | 4898.00 (3704.16) | 5996.11 (2089.00) | 7462.54 (5067.40) | 0.381 (0.11) |
| **RIO_LLMS** | 4451.10 (2747.50) | 6089.50 (3246.25) | 5813.20 (2816.20) | 0.451 (0.22) |
| **LIO_RLES** | 5895.10 (3488.42) | 7320.32 (5532.42) | 11463.63 (8278.01) | 0.150 (0.23) |
| **LIO_LLES** | 5994.11 (4754.82) | 6798.22 (3626.05) | 7476.64 (2634.40) | 0.722 (0.12) |
| **LIO_RLMS** | 5419.32 (3770.35) | 6152.75 (2613.52) | 8336.03 (5924.81) | 0.356 (0.24) |
| **LIO_LLMS** | 4976.00 (2964.71) | 5912.81 (3359.35) | 6515.85 (3429.75) | 0.601 (0.05) |

**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; $\eta^2$, Partial Eta Squared; External Oblique, EO; Internal Oblique, IO; Lumbar Erector Spinals, LES; Lumbar Multifidus, LMS; R, Right Side; L, Left Side

* Significant difference between AEP vs. CG, p = 0.026

** Significant difference between AEP vs. CG, p = 0.046

**Table 3. b.** Mean (SD) of trunk muscles' amplitudes and cocontraction indices (expressed as %sub-MVC) at minute 60 per group (N=30)

| Muscle | Minute 60 | | | p-value ($\eta^2$) |
|---|---|---|---|---|
| | Healthy Controls | AEP | FP | |
| **RIO** | 0.30 (0.23) | 0.42 (0.27) | 0.60 (0.53) | 0.231 (0.06) |
| **REO** | 0.80 (0.50) | 0.38 (0.19) | 0.61 (0.32) | 0.050 (0.20)** |
| **RLMS** | 0.22 (0.13) | 0.20 (0.06) | 0.21 (0.14) | 0.916 (0.02) |
| **RLES** | 0.26 (0.14) | 0.23 (0.09) | 0.33 (0.16) | 0.255 (0.22) |
| **REO_RLES** | 10141.57 (5877.40) | 10175.43 (4954.59) | 12991.62 (8348.86) | 0.587 (0.19) |
| **REO_LLES** | 8354.55 (4653.14) | 7114.08 (2099.35) | 8092.81 (4071.68) | 0.763 (0.04) |
| **REO_RLMS** | 8692.47 (5177.68) | 7877.77 (1987.67) | 8393.52 (6951.45) | 0.940 (0.26) |
| **REO_LLMS** | 6406.67 (3186.49) | 6989.27 (3504.45) | 8240.52 (4917.23) | 0.607 (0.18) |
| **LEO_RLES** | 9227.03 (4377.21) | 9358.80 (5747.70) | 13090.03 (7252.26) | 0.328 (0.03) |
| **LEO_LLES** | 8548.92 (4783.38) | 6644.17 (2421.34) | 7892.85 (3694.08) | 0.559 (0.17) |
| **LEO_RLMS** | 9065.99 (5834.36) | 7472.53 (3193.57) | 8013.14 (5979.72) | 0.801 (0.23) |
| **LEO_LLMS** | 6772.86 (3009.08) | 6553.63 (3533.80) | 8266.40 (4810.54) | 0.615 (0.24) |
| **RIO_RLES** | 6401.17 (4730.74) | 8861.17 (4011.01) | 11271.52 (8279.81) | 0.229 (0.12) |
| **RIO_LLES** | 6044.51 (4836.05) | 6365.63 (1853.47) | 7280.28 (4276.82) | 0.794 (0.21) |
| **RIO_RLMS** | 6192.85 (5281.91) | 6986.42 (1665.63) | 6910.75 (6065.44) | 0.921 (0.06) |
| **RIO_LLMS** | 5184.17 (3348.71) | 6761.86 (3625.68) | 6981.87 (4704.91) | 0.558 (0.19) |
| **LIO_RLES** | 7520.74 (5224.81) | 8405.51 (4707.80) | 11258.85 (7921.14) | 0.414 (0.21) |
| **LIO_LLES** | 7102.00 (5818.29) | 6210.12 (2061.92) | 7723.20 (4304.00) | 0.778 (0.07) |
| **LIO_RLMS** | 7231.42 (5298.66) | 6680.93 (2149.38) | 7629.40 (6360.20) | 0.922 (0.22) |
| **LIO_LLMS** | 5828.98 (3425.38) | 6237.75 (3333.80) | 7708.22 (4822.60) | 0.576 (0.14) |

**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; $\eta^2$, Partial Eta Squared; External Oblique, EO; Internal Oblique, IO; Lumbar Erector Spinals, LES; Lumbar Multifidus, LMS; R, Right Side; L, Left Side

* Significant difference between AEP vs. CG, p = 0.026

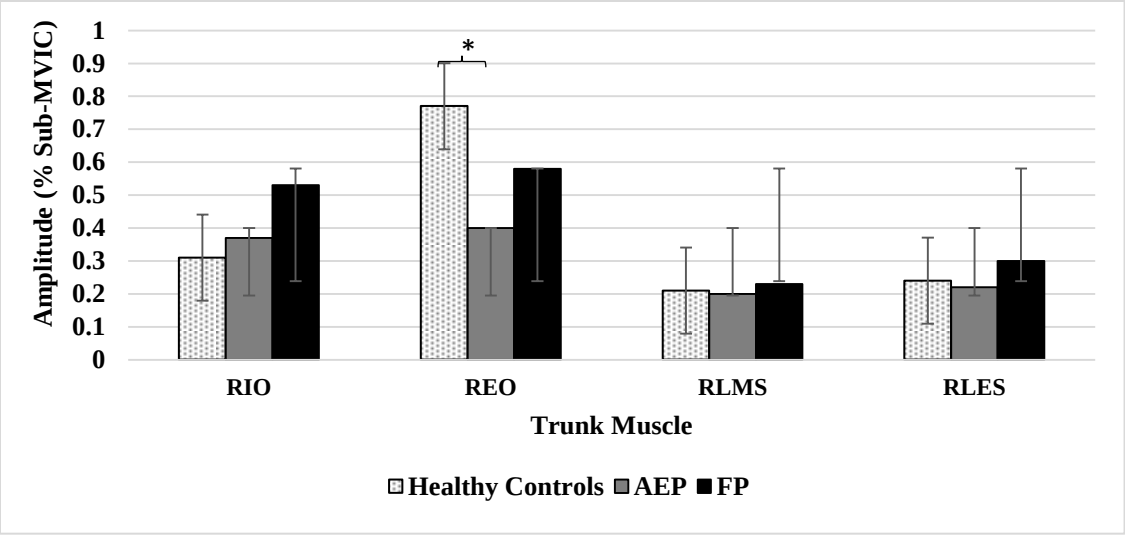
** Significant difference between AEP vs. CG, p = 0.046

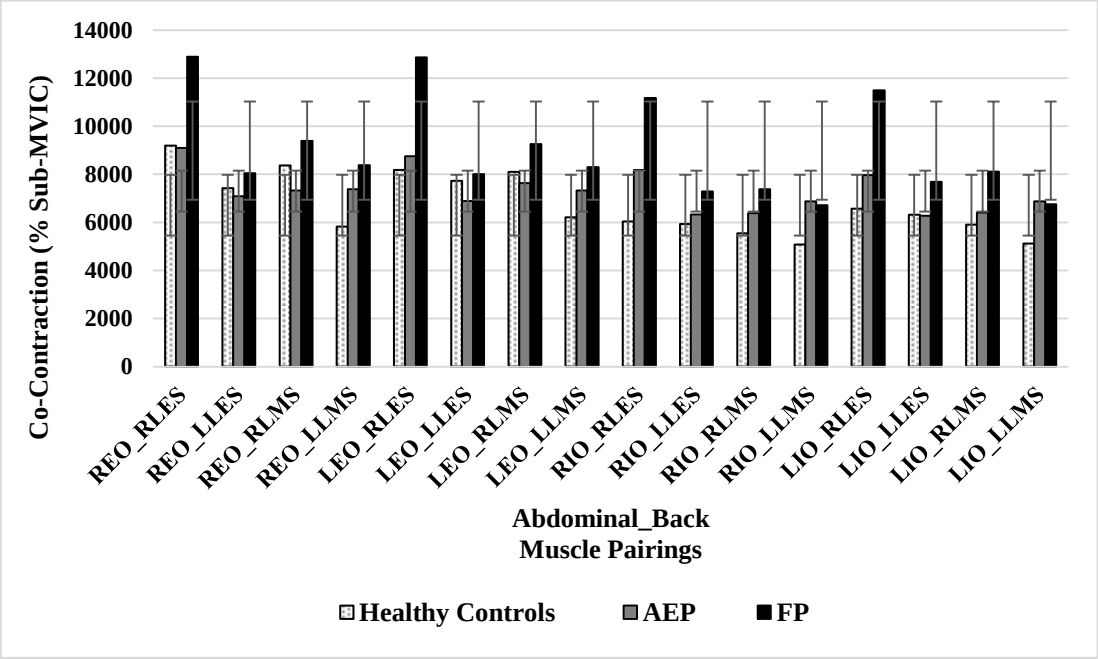
### *Secondary Analysis*

The differences in muscle activation amplitudes among all study groups over the entire 1-hour sitting are shown in **Figure 1a**. There was a significant difference in mean REO activation amplitude among the three study groups over the entire 1-hour sitting ($p=0.037$, $\eta^2= 0.22$). Bonferroni post hoc comparisons revealed that the difference in mean REO was significant between the AEP subgroup and healthy controls ($0.39\pm0.17$ vs. $0.77\pm0.42$, $p=0.033$). However, there was no significant difference in mean REO activation between FP and healthy controls ($p=0.52$).

No significant differences in mean activation amplitudes were found for the other muscle groups ($p>0.05$) as well as, no significate differences were noted for muscle pairings CCIs over the entire 1-hour sitting period ($p<0.05$). **Refer to Figure 1b.**

**a.** Mean (SD) of trunk muscles a. amplitudes (expressed as %sub-MVC) over the 1-hour sitting period by study group (N=30)






**b.** Mean (SD) of trunk muscles cocontraction indices (expressed as %sub-MVC) over the 1-hour sitting period by study group (N=30)

**Figure 1.** Mean (SD) of trunk muscles a. amplitudes, and b. cocontraction indices (expressed as %sub-MVC) over the 1-hour sitting period by study group (N=30)

**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; $\eta^2$, Partial Eta Squared; External Oblique, EO; Internal Oblique, IO; Lumbar Erector Spinals, LES; Lumbar Multifidus, LMS; R, Right Side; L, Left Side

*Significant difference between groups ($p \leq 0.05$)

### *Third Analysis*

**Figure 2.** shows the average pain scores of all groups over the 1-hour sitting. Results from the mixed factorial analysis showed a significant group by time interaction effect for pain ($p<0.001$, $\eta^2=0.47$). Results of the one-way ANOVA indicated that the difference in the amount of change from baseline was significant among the three groups at all time periods ($p<0.001$). Specifically, Bonferroni post hoc comparisons revealed that both pain subgroups significantly differed from healthy controls at all time periods ($p<0.05$). However, during the first 30 minutes of sitting, pain subgroups did not differ from each other, whereby, during the last 30 minutes, mean pain was significantly different between FP and AEP subgroups ($p<0.01$). Namely, the FP subgroup reported a significant increase in pain scores compared to the AEP subgroup at minute 40 ($38.1\pm15.0$ vs. $20.0\pm9.5$, $p=0.002$), minute 50 ($45.5\pm18.3$ vs. $24.5\pm16.1$, $p=0.007$), and minute 60 ($49.2\pm16.8$ vs. $27.4\pm19.7$, $p=0.009$). Similar results were found when adding pain at baseline as a covariate. The level of pain reported by the subgroups increased significantly over time ($p<0.001$, $\eta^2=0.80$ for FP and $\eta^2=0.44$ for AEP), whereby the pain peaked towards the end of the sitting period and increased significantly from baseline after 20 minutes of the sitting period (FP, $p<0.01$ and AEP, $p<0.05$).

There was no significant group by time interaction effect or over time change for all muscles' activity (amplitudes or CCIs) ($p>0.05$).

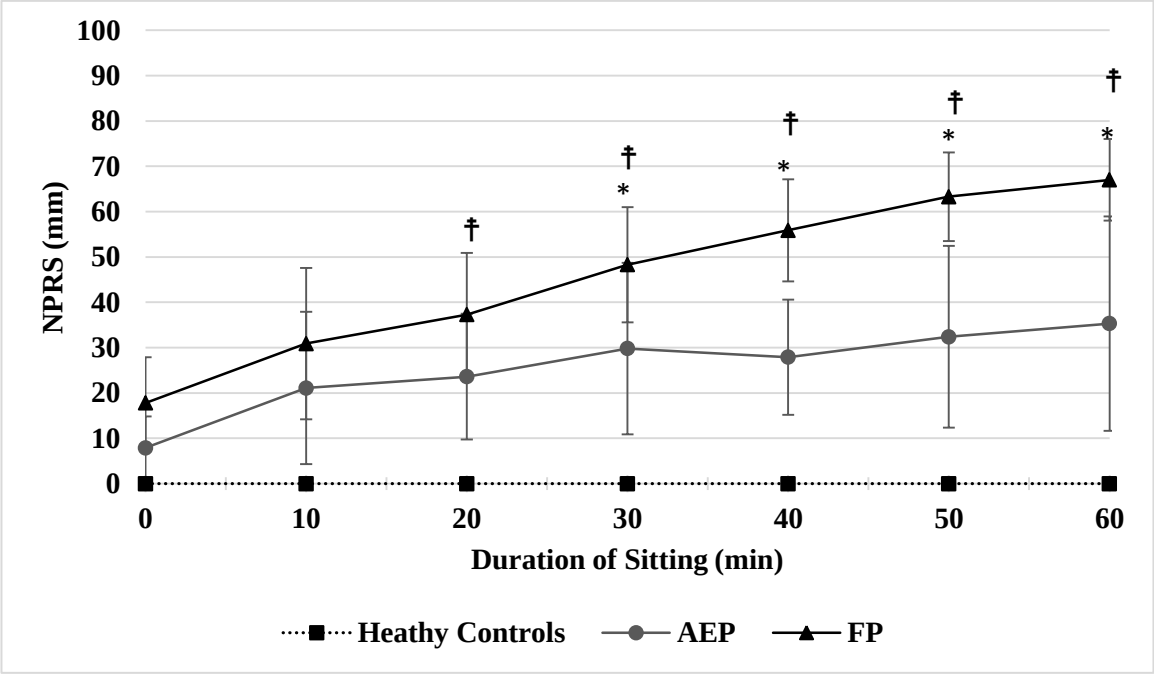



**Figure 2.** NPRS scores (SD) over the 1-hour sitting per group (N=30).
**Abbreviation:** FP, Flexion Pattern; AEP, Active Extension Pattern; NPRS, Numeric Pain Rating Scale
*Significant difference between pain subgroups; †Significant difference from Baseline NPRS ($p \leq 0.05$)

## Discussion

### *Summary of the findings*

The present study aimed to investigate trunk muscle activities (amplitudes and coactivations) among two commonly studied MCI subgroups (FP and AEP) compared to healthy controls, and their role on pain provocation over 1-hour of prolonged sitting period. Contrary to the hypothesis of this study, the results showed that all study groups presented with no significantly distinctive trunk muscles' activities at the beginning of sitting nor did they change overtime when pain increased to a significant level. Both MCI subgroups reported a similarly significant increase in pain through mid-sitting. However, after mid-sitting, both subgroups significantly differed from each other. This study's findings suggest no causative and/ or adaptive mechanisms for trunk muscles' activity on back pain development overtime.

### *Muscles' Activities (amplitudes and co-contraction indices)*

In the present study, all groups did not significantly differ from one another in truck muscles' activities at any point of time across the 1-hour sitting period. The muscle activities were similar among MCI subgroups at baseline, when pain was at its lowest level, and did not change even after pain provocation by means of prolonged sitting. This is in line with previous research studies in which trunk muscle activities were similar among pooled NSCLBP subjects compared to healthy controls [12, 16]. There are several possible explanations as to why muscle activities were not different, nor did they change overtime in the current study. First, the degrees of hip flexion play a significant role in trunk muscle activity, in fact, lower muscle activities were noted at lesser degrees of hip

flexion for both NSCLBP and healthy controls [14, 16]. Although all subjects sat on the same standard office chair, with hip angle relatively at 90 degrees, the lack of precise kinematic data regarding hip flexion might have led to the lack of significant differences in muscle activities among the study groups. Second, the subjects in the current study exhibited low levels of functional disability (mean RMDI for both MCI subgroups of 6.6 ±1.7) compared to previous studies. MCI subgroups with elevated functional disability are reported to have faulty muscular patterns during sitting when compared to healthy individuals and those with more disabling NSCLBP [13, 14, 31] . Therefore, this may explain why trunk muscle activities of MCI subgroups were not different from each other or their controls. Third, the trunk muscle activities and pain were only recorded for 1-hour of sitting with limited data on whether such period of time would result in meaningful differences between subjects with and without NSCLBP. Thus, the lack of differences in muscle activities might occur if the subject sat for prolonged sitting periods. Finally, a "muscle spasm model" has been identified among MCI subgroups and differences in muscle activities were established in previous studies, the current study was not able to support this notion. Previous research reported that the FP subgroup presented with increased abdominal muscle activities [12, 15, 35], whereas the AEP subgroup showed increased back muscle activities [11, 12, 14, 15]. However, few research studies have examined the effect of pain on truck muscle activities, or vice versa, using the co-contraction indices, as an indicator of the motor control response. CCIs are used to quantify the degree of spatial and temporal EMG data for a pair of muscle groups over a specific number of time points allowing a more objective measurement of the muscular patterns among study's groups [17, 21]. For instance,

Schinkel-Ivy, Nairn [17] reported that during 2 hours of sitting, low back pain developers displayed higher levels of cocontraction of trunk muscles than non-pain developers [17]. This co-contraction tended to increase over time and was directly associated with pain development. The authors suggested that such relationship could indicate a causative mechanism to pain development or an adaptive motor response in attempt to ease the pain [17]. Although the present study did not directly aim to examine the relationship between pain development and trunk muscle indices, the findings of this study revealed their inconsistent role on pain provocation overtime. Subclassification of NSCLBP in this study might have "washed out the effect" [12, 36] of muscle activities, and thus resulted in the prominence of similarities among the study's groups. This may support the notion that the change in muscle activities plays limited role on pain development overtime [16], and that when NSCLBP subgroups are classified based on the differences on muscle activities, the findings are highly variable. Therefore, examining other factors that might contribute to the driving mechanisms of back-pain disorder such as lumbosacral kinematics might assist in further validation of NSCLBP subgrouping models.

### *Study Limitations*

As highlighted, the levels of hip flexion were not measured in the present study, which might have omitted their influence in muscle activities. In addition, the sitting period was only monitored for an hour, which might not have provided a thorough understanding of how MCI subgroups operates during their daily office tasks that extends beyond an hour. However, due to a) the likelihood of experiencing LBP by these subgroups over a single hour, and b) the logistic of the testing which was used to limit the

sitting period to an hour to avoid unacceptable pain aggravation. Furthermore, the association between pain levels and muscle activities were not analyzed, thus we recommend that a correlation analysis be performed in order to examine this relationship. Also, sitting posture might not be challenging enough for the AEP subgroup to provoke pain due to its flexed nature [11, 18, 31], however, in the present study the AEP subgroup reported an increase in pain level over the 1-hour sitting. This can be attributed to the static loading at the lumbosacral spine associated with the prolonged sitting period making it a provocative means to aggravate pain in both studied MCI subgroups. Although the FP and AEP disorders are motor control deficits, future studies are warranted to monitor regional and segmental spine postures in conjunction with muscle activities overtime.

Another limited in this study was the small sample size included. A total of only 30 were enrolled in this study. A priori power analysis revealed that the power based on this sample was 0.80 with large effect size ($\eta^2 = 0.26$). However, the effect sizes reported in this study were less than 0.26. It is possible that we were not able to identify significant differences in muscle activities between the study groups due to the small sample size with the estimated large effect size. Thus, we recommend conducting further studies with a larger sample size to enhance the generalizability of the study's findings. Lastly, caution should be taken when interpreting the study's findings, since it is known that low functional disabilities among MCI subgroups had an effect on muscle activities [31]. We recommend that future studies to investigate muscle activities in people with more disabling LBP as it might illuminate the difference in muscle activities among the studied groups.

### *Clinical Implications*

The findings of the present study highlighted the similarities in trunk muscle activities among NSCLBP patients related to MCI and healthy controls while sitting for an extended period of time. Recognizing these muscular patterns and their limited contributions to pain development might enhance the application of the classification-based cognitive functional therapy (CB-CFT) [37], mainly in FP and AEP subgroups. A postural biofeedback rather than muscular activations' training to facilitate proper lumbosacral kinematics might be relevant in spine rehabilitation [37, 38]. Finally, incorporation of these findings to intervention approaches might advance NSCLBP management.

## Conclusion

The results of this study showed that subjects with and without NSCLBP presented with similar muscular patterns, and the significant increase in pain among the NSCLBP subgroups related to MCI over the 1-hour sitting might not be attributed to these muscular patterns. Incorporating these findings into treatment strategies might assist in reducing back pain among MCI subgroups.

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# CHAPTER FOUR

# DISCUSSION

## Lumbosacral Kinematics and Muscle Activities in Non-specific Chronic Low Back Pain Subgroups

### *Summary of the lumbosacral kinematics' findings*

The present study aimed to investigate spine postural behaviors among two commonly studied MCI subgroups (FP and AEP) compared to heathy controls, and their role on pain development during a 1-hour of prolonged sitting. The results of this study showed that all study groups presented with significantly distinctive postural behaviors at the beginning and at the end of the sitting period. Only the MCI subgroups, however, showed significant deterioration in the lumbosacral kinematics and pain levels overtime. The direction of deterioration in lumbosacral kinematics over the 1-hour sitting period occurred in the direction of the motor control impairment (kyphosis for FP or lordosis for the AEP subgroup). Interestingly, both MCI subgroups reported a similarly significant increase in pain through mid-sitting. However, after mid-sitting, the AEP subgroup displayed a significantly reversed decrease in the lordotic posture which was accompanied by much less increase in pain level compared to the FP subgroup. The findings of this study suggest a possible association between the lumbosacral postures and pain development overtime.

### FP subgroup

In the present study, the FP subgroup exhibited an increased kyphotic presentation of the lower lumbosacral region over the entire 1-hour of sitting period as compared to the other groups. Interestingly, the observed differences in kinematics did not only appear

after the onset of increased pain, instead, they were initially present at baseline, which may further suggest an inherent postural behaviors in the FP subgroup that predisposed them to pain (15, 33). In addition, these behaviors continued to deteriorate in the direction of flexion throughout testing, which could imply an overtime loss of the motor control of the moving lower lumbosacral segments in the direction of pain provocation (9) contributing to the increased pain overtime. Furthermore, over the 1-hour sitting period, FP subgroup did not show any attempt to produce a positional alteration in order to reduce their pain, instead, they maintained a directional increase of the lower lumbosacral flexion. This might further support the presence of a "neutral spinal position deficit" in which they underestimated the neutral position of lower lumbar by adopting a kyphotic posture (34) throughout the sitting period resulting in pain provocation. Lastly, the FP subgroup spent the majority of their sitting, in general, at end-range flexion posture. Maintaining an end-range posture from the beginning through the end of sitting period, as shown clearly in the present study, was accompanied by a significant increase in pain overtime. Specifically, the pain increased to statistically significant level from the beginning of the sitting period after >20 minutes and remained significant for the rest of the sitting period. This could suggest that the exhibited flexion end-range behavior might have caused a progressive increase of the lower lumbar strain in the FP subgroup, disrupting the physiological distribution of spinal loading in lumbosacral structures, leading to the incremental increase in pain over the sitting period (15, 33).

Similar to these findings, previous studies reported a greater posterior sacral tilt (15, 16) and flexion of the lower lumbar angle (15) in the FP subgroup during a 5-second, 1-hour sitting (17), and field cycling (33). Similar to the findings of the present study, the

cited studies attributed pain increase to the sustain extreme flexion posture in the FP subgroup (17, 20, 35) secondary to: a) inherent motor control impairments in the direction of flexion, b) proprioceptive alterations of spine structures (34), and c) restricted lumbosacral range of movement toward the opposite direction of the motor control deficits (15). Lastly, it is important to note that the present study determined the direction of the postural control impairment over a 1-hour sitting period and identified its influence in pain increase within the FP subgroup. This could further clarify the underlying mechanism of pain development and eventually assist in the development of customized chairs or biofeedback training approaches into clinical practice to address these postural faults adopted by this subgroup.

**AEP subgroup**

In contrast, the AEP subgroup exhibited an increased lordotic posture of the lower lumbosacral region over the entire 1-hour of sitting period as compared to the FP and healthy controls. Similar to the FP subgroup, the observed differences in kinematics were present at baseline and evidently appeared after the onset of pain, which again may further support the presence of the inherent postural behaviors in the AEP subgroup (15, 33). In the same manner to the FP subgroup, the observed postural behaviors continued to deteriorate in the direction of extension but only throughout the early phase of sitting (<40 minutes), which may suggest an overtime loss of the motor control of the lower lumbosacral segments (9) contributing to the increased pain at the early phases of sitting. However, after 40 minutes of sitting, the AEP subgroup showed some positional alteration toward “neutral” which could be interpreted as an adaptive approach to reduce/control their pain level. This finding might further support that the AEP subgroup

possibly had a “neutral spinal position deficit” only at the early phase of sitting period, in which they underestimated the neutral position of lower lumbar by adopting a lordotic posture (34) resulting in pain provocation. However, less positional deficits were observed after 40 minutes of sitting as the AEP subgroup moved toward “neutral”. Lastly, the AEP subgroup spent most of their early sitting time at end-range extension. Sustaining an end-range postures from the beginning through the mid-point of sitting period was associated with a significant increase in pain. Specifically, the pain increased to statistically significant level from the beginning of the sitting period after >20 minutes through the end. Although, the pain after 40 minutes was significantly different from baseline, it was slightly lower compared to the FP subgroup. The initial lordotic behavior over the 1-hour sitting may contribute to the increased extension compressive force, developed by sustained lumbar extension and possible muscle fatigue, in posterior spinal structures resulting in development of pain (15). However, the later correction of the postural faults toward neutral might explain the noted reduction in pain level toward the end of the sitting period in this MCI subgroup.

Similar to these findings, previous studies reported a greater anterior sacral tilt (15, 16) and extension of the lower lumbar angle (15) in the AEP subgroup during 5-second sitting, 10-minute (20), and functional tasks (11). Although sitting in a standard chair might have promoted lower lumbar flexion in the AEP subgroup (20), ironically they assumed hyperextension postures away from the neutral spectrum resulting in pain increase in the first 40 minutes of sitting. However, toward the end of sitting period they assumed more of a neutral posture which was associated with relatively lesser back pain compared to the FP subgroup. The initial lordotic posture could be explained by reduced

ability of the AEP subgroup to relax their paraspinal muscles which in return might have minimizing their ability to tilt their pelvis posteriorly (13, 20). Unfortunately, the lack of muscle activation data hindered the ability to confirm this notion. The latter "neutral posture" is in line with the previously reported findings by Curran et al. (2014) [20], in which the AEP subgroup reported greater back discomfort when they sat on a forward inclined seatpan, but the pain was lower while sitting on a flat seatpan chair. The findings from this study could imply that the positions of lumbosacral region in the AEP subgroup were maybe related to pain alterations.

### *Summary of the muscles activity's findings*

The present study aimed to investigate trunk muscle activities (amplitudes and coactivations) among two commonly studied MCI subgroups (FP and AEP) compared to healthy controls, and their role on pain provocation over 1-hour of prolonged sitting period. Contrary to the hypothesis of this study, the results showed that all study groups presented with no significantly distinctive trunk muscles' activities at the beginning of sitting nor did they change overtime when pain increased to a significant level. Both MCI subgroups reported a similarly significant increase in pain through mid-sitting. However, after mid-sitting, both subgroups significantly differed from each other. This study's findings suggest no causative and/ or adaptive mechanisms for trunk muscles' activity on back pain development overtime.

**Muscles' Activities (amplitudes and co-contraction indices)**

In the present study, all groups did not significantly differ from one another in truck muscles' activities at any point of time across the 1-hour sitting period. The muscle activities were similar among MCI subgroups at baseline, when pain was at its lowest level, and did not change even after pain provocation by means of prolonged sitting. This is in line with previous research studies in which trunk muscle activities were similar among pooled NSCLBP subjects compared to healthy controls [12, 16]. There are several possible explanations as to why muscle activities were not different, nor did they change overtime in the current study. First, the degrees of hip flexion play a significant role in trunk muscle activity, in fact, lower muscle activities were noted at lesser degrees of hip flexion for both NSCLBP and healthy controls [14, 16]. Although all subjects sat on the same standard office chair, with hip angle relatively at 90 degrees, the lack of precise kinematic data regarding hip flexion might have led to the lack of significant differences in muscle activities among the study groups. Second, the subjects in the current study exhibited low levels of functional disability (mean RMDI for both MCI subgroups of 6.6 ±1.7) compared to previous studies. MCI subgroups with elevated functional disability are reported to have faulty muscular patterns during sitting when compared to healthy individuals and those with more disabling NSCLBP [13, 14, 31] . Therefore, this may explain why trunk muscle activities of MCI subgroups were not different from each other or their controls. Third, the trunk muscle activities and pain were only recorded for 1-hour of sitting with limited data on whether such period of time would result in meaningful differences between subjects with and without NSCLBP. Thus, the lack of differences in muscle activities might occur if the subject sat for prolonged sitting

periods. Finally, a "muscle spasm model" has been identified among MCI subgroups and differences in muscle activities were established in previous studies, the current study was not able to support this notion. Previous research reported that the FP subgroup presented with increased abdominal muscle activities [12, 15, 35], whereas the AEP subgroup showed increased back muscle activities [11, 12, 14, 15]. However, few research studies have examined the effect of pain on truck muscle activities, or vice versa, using the co-contraction indices, as an indicator of the motor control response. CCIs are used to quantify the degree of spatial and temporal EMG data for a pair of muscle groups over a specific number of time points allowing a more objective measurement of the muscular patterns among study's groups [17, 21]. For instance, Schinkel-Ivy, Nairn [17] reported that during 2 hours of sitting, low back pain developers displayed higher levels of cocontraction of trunk muscles than non-pain developers [17]. This co-contraction tended to increase over time and was directly associated with pain development. The authors suggested that such relationship could indicate a causative mechanism to pain development or an adaptive motor response in attempt to ease the pain [17]. Although the present study did not directly aim to examine the relationship between pain development and trunk muscle indices, the findings of this study revealed their inconsistent role on pain provocation overtime. Subclassification of NSCLBP in this study might have "washed out the effect" [12, 36] of muscle activities, and thus resulted in the prominence of similarities among the study's groups. This may support the notion that the change in muscle activities plays limited role on pain development overtime [16], and that when NSCLBP subgroups are classified based on the differences on muscle activities, the findings are highly variable. Therefore, examining other factors that might

contribute to the driving mechanisms of back-pain disorder such as lumbosacral kinematics might assist in further validation of NSCLBP subgrouping models.

### *Clinical Implications*

The findings of the present study highlight the postural behaviors that NSCLBP patients with MCI display while sitting for an extended period of time. Identifying these behaviors and their contributions to pain development might refine the application of the classification-based cognitive functional therapy (CB-CFT) (14) in FP and AEP subgroups. A postural biofeedback training to facilitate proper lumbosacral kinematics away from the end-range sitting postures, could be relevant in spine rehabilitation for these subgroups (14, 36). Also, incorporation the findings to intervention approaches for these subgroups might advance NSCLBP management. For example, ergonomic recommendations regarding the use of a lumbar roll for the FP subgroup and a declined seatpan for the AEP subgroup might assist in pain reduction among the MCI subgroups. Also, this study highlighted the similarities in trunk muscle activities among NSCLBP patients related to MCI and healthy controls while sitting for an extended period of time. Recognizing these muscular patterns and their limited contributions to pain development might improve NSCLBP management. For instance, a postural biofeedback rather than muscular activations' training to facilitate proper lumbosacral kinematics might be relevant in spine rehabilitation [37, 38].

### *Conclusion*

The results of this study showed that both MCI subgroups presented with similar muscular patterns, but distinctive underlying maladaptive postural patterns. The

significant increase in pain over the 1-hour sitting might not be only attributed to the inherent maladaptive postures, also it may be related to the directional deterioration in lumbosacral postures overtime. Incorporating these findings into treatment strategies might assist in reducing sitting back pain among MCI subgroups.

### *Study Limitations and Future Recommendation*

The sitting period was only monitored for an hour, which might not have provided a thorough understanding of how MCI subgroups operates during their daily office tasks that extends beyond an hour. However, due to a) the likelihood of experiencing LBP by these subgroups over a single hour, and b) the logistic of the testing, it was intended to limit the sitting period to an hour to avoid unacceptable pain aggravation. Furthermore, the association between pain levels and lumbosacral kinematics were not analyzed, however, the purpose of this study was to establish the differences among the MCI subgroups in lumbosacral kinematics and muscle activation prior to the onset of pain increase and at the highest levels of pain over an hour of sitting. Also, sitting posture might not be challenging enough for the AEP subgroup to produce pain due to its flexed nature (15-17), however, in the present study the AEP subgroup reported an increase in pain level over the 1-hour sitting. This can be attributed to the static loading at the lumbosacral spine associated with the prolonged sitting period making it a provocative means to aggravate pain in both studied MCI subgroups. Although the FP and AEP disorders are sagittal plane motor control deficits, so the two-dimensional inclinometer would sufficiently capture the deviations from the sagittal plane, future studies are warranted to monitor regional and segmental spine postures in the three planes of

movements overtime. Unfortunately, this was not available for the present study. In addition, the levels of hip flexion were not measured in the present study, which might have omitted their influence in muscle activities.

Another limited in for the muscle activation study was the small sample size included. A total of only 30 were enrolled in this study. A priori power analysis revealed that the power based on this sample was 0.80 with large effect size ($\eta^2 = 0.26$). However, the effect sizes reported in this study were less than 0.26. It is possible that we were not able to identify significant differences in muscle activities between the study groups due to the small sample size with the estimated large effect size. Thus, we recommend conducting further studies with a larger sample size to enhance the generalizability of the study's findings. Lastly, caution should be taken when interpreting the study's findings, since it is known that low functional disabilities among MCI subgroups had an effect on muscle activities [31]. We recommend that future studies to investigate muscle activities in people with more disabling LBP as it might illuminate the difference in muscle activities among the studied groups.

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# CHAPTER FIVE

## Appendices

## Informed Consent Form

## INFORMED CONSENT

**TITLE:** **NON-SPECIFIC CHRONIC LOW BACK PAIN: EXAMINING TRUNK MUSCLE ACTIVATION AND KINEMATICS, AND THEIR ASSOCIATIONS WITH PAIN DEVELOPMENT DURING PROLONGED SITTING IN SUBGROUPS OF MOTOR CONTROL IMPAIRMENT**

**SPONSOR:** **Department of Physical Therapy, Loma Linda University**

**PRINCIPAL INVESTIGATOR:** **Everett Lohman, III, D.Sc., P.T., OCS, Professor, Department of Physical Therapy**

### WHY IS THIS STUDY BEING DONE?

The purpose of this graduate student research is to compare the differences in spinal curvatures and trunk muscle activity among subgroups of Non-specific Chronic Low Back Pain (NSCLBP) and healthy individuals, and their associations with pain experience during prolonged sitting.

You are invited to participate in this research study because you are an adult between the age of 18 to 65 years old, and healthy; or you have had Low Back Pain (LBP) that has lasted for more than 3 months until the present, you have pain localized to the lower back, you have not been diagnosed with specific LBP such as cauda equina syndrome or inflammatory disease; you will be excluded if you have had previous spinal surgery, you are pregnant (self-reported) at the time of the study, 6 months postpartum, or you recently underwent a period of lower back and abdominal muscles strengthening program.

Approximately 80 subjects will participate in this study. The study will last one session. It will require 1.5 – 2 hours on the day of data collection.

### HOW WILL I BE INVOLVED?

- You will complete the informed consent at Nichol Hall, Room Number A620 or A640
- You will complete a patient information form and undergo a back examination to determine your eligibility for the study

- You will be then given a hard copy of self-reported questionnaires to complete either during the session or at your best convenience within a one-week window from the initial session window (a self-addressed stamped envelope to return the completed questionnaires to the principal investigator will be provided)
- You will need to remove your top shirt for males or wear sport bra for females to expose your lower abdominal and back area to place the electrodes and markers (a backless vest top, changing room and privacy curtains will be provided if needed)
- Small areas of the skin might be shaved (if necessary; 2x2 cm) and cleaned with alcohol prior to the electrodes and markers placement
- A series of muscle contraction of standardized protocols (3 trials of legs lifting while lying on the back and then on stomach) will be performed prior to carrying out the actual sitting protocol with 1 minute of rest between trials
- You will be then provided with a standard office workstation setup and will spend one hour sitting reading passages from selected sources on the monitor.
- While sitting, trunk muscle activations will be recorded. This will involve placement of electromyography electrodes (noninvasive device that is used to measure the muscle activation) onto the surface of the skin to record muscle activity during sitting protocol.
- While sitting, spinal curvatures will be recorded. This will involve placement of movement sensors (noninvasive device that is used to measure joint position) onto the surface of the skin to record spinal segment movement during sitting protocol.
- Pain level using a self-reported scale prior to beginning the one-hour sitting protocol, and every 10 minutes throughout the 1-hour sitting protocol (total of seven readings)

**WHAT ARE THE REASONABLY FORESEEABLE RISKS OR DISCOMFORTS I MIGHT HAVE?**

Some of the testing procedures will require you to sit for a full hour in front of the monitor. This will put you at minimal risk to develop back discomfort. Also, you may feel fatigued or bored due to testing procedures. This will be minimized by employing rest period and reporting any adverse effect or concerns about the procedures to the investigator. Additionally, there might be a risk of embarrassment that will be minimized by private screen area of curtains. Lastly, there is also a minimal risk of breach of confidentiality. However, all records and research materials that identify you will be held confidential. Any published document resulting from this study will not disclose your identity without your permission. Information identifying you will only be available to the study personnel. All subjects will be identified with a numeric code. The identification key will not be destroyed and will be kept in a cabinet in a locked room.

**WILL THERE BE ANY BENEFIT TO ME OR OTHERS?**

Although you may not personally benefit from this study, your participation may help practitioners better understand movements and muscle activation patterns in low back pain patients during prolonged sitting. This will benefit other subjects with similar conditions in the future and will advance the research in this particular area.

**WHAT ARE MY RIGHTS AS A SUBJECT?**

Your participation in this study is entirely voluntary. You may refuse to participate or withdraw once the study has started. Your decision whether or not to participate or terminate at any time will not affect your future medical standing with the researchers. You do not give up any legal rights by participating in this study. If at any time you feel uncomfortable, you may refuse to answer questions.

**WHAT COSTS ARE INVLOVED?**

There is no cost to you for your participation in this study beyond the time involved to participate. You will be responsible for your own travel to and from the research lab.

**WILL I BE PAID TO PARTICIPATE IN THIS STUDY?**

You will receive a $50 gift card for completion of the study in full. In order to receive such payment, you may be asked to provide your name and your Loma Linda University ID number if you are a student or an employee.

**WHO DO I CALL IF I HAVE QUESTIONS?**

If you have any concerns or questions regarding this research, please contact Everett Lohman, III, D.Sc., P.T., OCS at elohman@llu.edu or (909) 558-4632 or Ext. 83171.

If you wish to contact an impartial third party not associated with this study regarding any questions about your rights or to report a complaint you may have about the study, you may contact the office of Patient Relations, Loma Linda University Medical Center, Loma Linda, CA 92354, phone (909) 558-4647, e-mail: patientrelation@llu.edu for information and assistance.

**SUBJECT'S STATEMENT OF CONSENT**

- I have read the contents of the consent form and have listened to the verbal explanation given by the investigator.
- My questions concerning this study have been answered to my satisfaction.
- Signing this consent document does not waive my rights nor does it release the investigators, institution or sponsors from their responsibilities.
- I may call Everett Lohman, III, D.Sc. during routine office hours at (909) 558-4632 or Ext. 83171 if I have additional questions or concerns.
- I hereby give voluntary consent to participate in this study.

I understand I will be given a copy of this consent form after signing it.

_____ _____

Signature of Subject Printed Name of Subject

_____
Date

**INVESTIGATOR'S STATEMENT**

I have reviewed the contents of this consent form with the person signing above. I have explained potential risks and benefits of the study.

_____ _____
Signature of Investigator Printed Name of Investigator

_____
Date

**Study Flyer**

LOMA LINDA UNIVERSITY
School of Allied Health Professions

# Research Opportunity

"Non-specific Chronic Low Back Pain: Examining Trunk Muscle Activation and Kinematics, and their Associations with Pain Development During Prolonged Sitting in Subgroups of Motor Control Impairment"

**PARTICIPANTS NEEDED**
**Loma Linda University**
**School of Allied Health Profession**

We are looking for volunteers for a graduate student research study.

- This graduate student study will be held on the LLU campus, Nichol Hall, Room A620 and A640.
- Your participation in this study will last for 1.5 -2 hours for one visit to undergo low back examination and measuring spinal muscle activity and curvatures during prolonged sitting
- Subject will receive a small gift.

You may qualify to participate in this study if you are between 18-65 years old and:

- Healthy; or you have
- ≥ 3 months low back pain (LBP), and
- Pain localized to the lower back, and
- Absence of "red flags" (specific causes of LBP such as cauda equina syndrome or inflammatory disease)

You may be excluded if:

- Previous spine surgery
- Pregnant at the time of the study or 6 months postpartum
- Recently undergone a period of motor control rehabilitation

Principal Investigator: Everett Lohman III, PT, DSc, OCS.
Sponsor: Loma Linda University- Department of Physical Therapy.

**For More Information Contact:** Graduate student investigator: Mansoor Alameri at: Malameri@llu.edu or Cell: (617)372-1744

**Subject Demographics Form**

LOMA LINDA UNIVERSITY
School of Allied Health Professions

*Data Collection Sheet*

*NON-SPECIFIC CHRONIC LOW BACK PAIN: EXAMINING TRUNK MUSCLE ACTIVATION AND KINEMATICS, AND THEIR ASSOCIATIONS WITH PAIN DEVELOPMENT DURING PROLONGED SITTING IN SUBGROUPS OF MOTOR CONTROL IMPAIRMENT*

# PATIENT'S INFORMATION FORM

Participant's ID: ……... Date: ……. /……/…….. Check-in Time: ……**:**

…….

Fist Name: ……. Last Name: …….

Age in Years: ……

Gender: M ☐ F ☐

Phone Number: **(**……**)** ……-…….

Email: ……**@**…….

Preferred Contact Method: Phone ☐ Email ☐

Hight: ……... (Feet Or Cm)

Weight: …… (Lbs. Or Kg)

***Please answer the following questions:***

**1.** Are you between 18 to 65 years' old? ***YES*** ***NO***

**2.** Have you had Low Back Pain that has lasted for more than 3 months? ***YES***

***NO***

**3.** In a scale of 100%, how long the issue of back pain has been present in the past 6 months?

- More than 50% of the time. ***YES NO***
- Less than 50% of the time. ***YES NO***

**4.** Have you had Low Back Pain of equal or greater than **2/10** in the past week? ***YES NO***

**4.** "Which situation describes your pain over the past 4 weeks the best?

- 100% of the pain in the low back. ***YES NO***
- 80% of the pain in the low back, but 20% on the leg/s. ***YES NO***
- More than 20% of the pain on my leg/s. ***YES NO***

**5.** Please complete the Roland Morris Disability Questionnaire (RMDQ); (**The principal investigator will calculate your score**)

- Did you score **equal or more than 5 points** in RMDQ? ***YES NO***

**6.** Have you had previous extensive spinal surgery (**greater than single-level fusion/instrumentation or discectomy**)? ***YES NO***

**7.** Have you had serious spinal pathology (**cancer, inflammatory arthropathy, acute vertebral fracture or cauda equine**)? ***YES NO***

**8.** Have you been diagnosed with neurological disease? ***YES NO***

**9.** Have you had a psychiatric history (**currently under the care of a mental health care provider or taking multiple psychiatric medications**)? ***YES NO***

**10.** Do you have widespread chronic syndrome (eg, Fibromyalgia)? ***YES NO***

**11.** In the past 6 months, has you undergone a period of motor control rehabilitation (**Core muscle strengthening program under the supervision of a Physical Therapist**)? ***YES NO***

***12.*** If you are a **female**, are you pregnant the time of the study or 6 months postpartum? ***YES NO***

**Numeric Pain Rating Scale (NPRS)**

## Patient ID:

Please mark your pain level ***in the past 24 hours*** where 0 represents "no pain" and 100 represents "worst pain imaginable":



**0** "No Pain"

**100** "Worst Pain imaginable"

**Numeric Pain Rating Scale (NPRS) for Overtime**

**Patient ID:**

Please mark your pain level ***at this moment*** where 0 represents "no pain" and 100 represents "worst pain imaginable":

**Recording number:**

Baseline 2nd 3rd 4th 5th 6th 7th

**0** **100**

"No Pain" "Worst Pain imaginable"

# The Ronald Morris Disability Questionnaire

The Roland-Morris Low Back Pain and Disability Questionnaire

Patient name: _____ File #_____ Date:_____

Please read instructions: When your back hurts, you may find it difficult to do some of the things you normally do. Mark only the sentences that describe you today.

- ☐ I stay at home most of the time because of my back.
- ☐ I change position frequently to try to get my back comfortable.
- ☐ I walk more slowly than usual because of my back.
- ☐ Because of my back, I am not doing any jobs that I usually do around the house.
- ☐ Because of my back, I use a handrail to get upstairs.
- ☐ Because of my back, I lie down to rest more often.
- ☐ Because of my back, I have to hold on to something to get out of an easy chair.
- ☐ Because of my back, I try to get other people to do things for me.
- ☐ I get dressed more slowly than usual because of my back.
- ☐ I only stand up for short periods of time because of my back.
- ☐ Because of my back, I try not to bend or kneel down.
- ☐ I find it difficult to get out of a chair because of my back.
- ☐ My back is painful almost all of the time.
- ☐ I find it difficult to turn over in bed because of my back.
- ☐ My appetite is not very good because of my back.
- ☐ I have trouble putting on my sock (or stockings) because of the pain in my back.
- ☐ I can only walk short distances because of my back pain.
- ☐ I sleep less well because of my back.
- ☐ Because of my back pain, I get dressed with the help of someone else.
- ☐ I sit down for most of the day because of my back.
- ☐ I avoid heavy jobs around the house because of my back.
- ☐ Because of back pain, I am more irritable and bad tempered with people than usual.
- ☐ Because of my back, I go upstairs more slowly than usual.
- ☐ I stay in bed most of the time because of my back.

Instructions:

1. The patient is instructed to put a mark next to each appropriate statement.

2. The total number of marked statements are added by the clinician. Unlike the authors of the Oswestry Disability Questionnaire, Roland and Morris did not provide descriptions of the varying degrees of disability (e.g., 40%-60% is severe disability).

3. Clinical improvement over time can be graded based on the analysis of serial questionnaire scores. If, for example, at the beginning of treatment, a patient's score was 12 and, at the conclusion of treatment, her score was 2 (10 points of improvement), we would calculate an 83% (10/12 x 100) improvement.

# Tampa Scale of Kinesophobia

## Tampa Scale for Kinesiophobia

(Miller , Kori and Todd 1991)

1 = strongly disagree
2 = disagree
3 = agree
4 = strongly agree

| | | | | |
|---|---|---|---|---|
| 1. I'm afraid that I might injury myself if I exercise | 1 | 2 | 3 | 4 |
| 2. If I were to try to overcome it, my pain would increase | 1 | 2 | 3 | 4 |
| 3. My body is telling me I have something dangerously wrong | 1 | 2 | 3 | 4 |
| 4. My pain would probably be relieved if I were to exercise | 1 | 2 | 3 | 4 |
| 5. People aren't taking my medical condition seriously enough | 1 | 2 | 3 | 4 |
| 6. My accident has put my body at risk for the rest of my life | 1 | 2 | 3 | 4 |
| 7. Pain always means I have injured my body | 1 | 2 | 3 | 4 |
| 8. Just because something aggravates my pain does not mean it is dangerous | 1 | 2 | 3 | 4 |
| 9. I am afraid that I might injure myself accidentally | 1 | 2 | 3 | 4 |
| 10. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening | 1 | 2 | 3 | 4 |
| 11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body | 1 | 2 | 3 | 4 |
| 12. Although my condition is painful, I would be better off if I were physically active | 1 | 2 | 3 | 4 |
| 13. Pain lets me know when to stop exercising so that I don't injure myself | 1 | 2 | 3 | 4 |
| 14. It's really not safe for a person with a condition like mine to be physically active | 1 | 2 | 3 | 4 |
| 15. I can't do all the things normal people do because it's too easy for me to get injured | 1 | 2 | 3 | 4 |
| 16. Even though something is causing me a lot of pain, I don't think it's actually dangerous | 1 | 2 | 3 | 4 |
| 17. No one should have to exercise when he/she is in pain | 1 | 2 | 3 | 4 |

Reprinted from:
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