Investigating biopsychosocial, methodological, and task-based determinants of inter-joint coordination in adults with chronic low back pain.

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This thesis is dedicated to the loving memory of my mother, Susan (1952-2020).

Thank you for everything.

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List of abbreviations

ANOVA: Analysis of variance

BMI: Body mass index

BPI: Brief pain inventory

CI: Confidence interval

CoP: Center of pressure

CRIR: Centre de recherche interdisciplinaire en réadaptation du Montréal métropolitain

CRP: Continuous relative phase

CSQ-Cat: Coping strategies questionnaire catastrophizing subscale

DAG: Directed acyclic graph

DP: Deviation phase

FABQ: Fear-avoidance belief questionnaire

FABQ-PA: Fear-avoidance belief questionnaire physical activity subscale

FABQ-W: Fear-avoidance belief questionnaire work subscale

FFD: Finger-floor distance

FRR: Flexion-relaxation ratio

IRSST: Institut de recherche Robert Sauvé en santé et sécurité du travail

LBP: Low back pain

LowLx: Lower lumbar (L3-S1)

MARP: Mean absolute relative phase

NPRS: Numeric pain rating scale

ODI: Oswestry disability index

OPPQ-REPAR: Ordre professionel de la physiothérapie du québec – réseau provinciale de recherche en adaptation-réadaptation

PASS: Pain and anxiety symptoms scale

PCS: Pain catastrophizing scale

PHODA: Photograph series of daily activities

PHODA-Sev: Photograph series of daily activities short electronic version

PRISMA: Preferred reporting items for systematic reviews and meta-analyses

RMSD: Root mean squared deviation

RMS: Root mean square

ROM: Range of motion

SD: Standard deviation

sEMG: Surface electromyography

SMD: Standardized mean difference

STS: Sit-to-stand-to-sit

TSK: Tampa scale for kinesiophobia

TSK-AA: Tampa scale for kinesiophobia activity avoidance subscale

TSK-SF: Tampa scale for kinesiophobia somatic focus subscale

UpLx: Upper lumbar (T12-L3)

Abstract

Pain may serve as a protective mechanism, prompting guarding of a region that is perceived to be injured or threatened. In people with low back pain (LBP), decreased spinal motion, tighter hip-spine coordination, and reduced variability are thought to reflect guarded motor behaviors. While these behaviors may cause abnormal tissue loading and complicate recovery, they are inconsistently observed in the literature and poorly understood. These inconsistencies might relate to (i) task-related considerations, (ii) biomechanical methodological procedures, and (iii) the influence of biopsychosocial variables. Better understanding of these relationships can fill knowledge gaps and help inform treatment strategies and future research. Therefore, the overarching objective of this thesis was to understand alterations in motor behaviors in people with LBP, by studying the determinants of spinal motion and hip-spine coordination. This overarching objective was achieved via four manuscripts.

First, a systematic review and meta-analysis summarized the relationship between motor behavior (i.e., spinal range of motion, trunk muscle activity, and coordination) and pain-related threat (via pain catastrophizing and pain-related fear), in adults with LBP (Chapter 3). This review searched 4 databases, and articles were screened and rated for quality by separate reviewers. Twenty-one studies were retained for 5 meta-analyses, which were performed using the random-effects model. A small effect associated greater pain-related threat with guarded motor behavior (i.e., decreased spinal range of motion, and greater trunk muscle activity) during flexion-based tasks, but not consistently during other movements. A lack of available studies precluded the analysis of the relationship between pain-related threat and coordination.

Next, we compared hip-spine coordination and variability, during a novel task, in people with (n = 16) and without LBP (n = 21) (Chapter 4). We used previously collected lumbo-pelvic

kinematic data from a sit-to-stand-to-sit task and performed Continuous Relative Phase (CRP) analyses using a Hilbert transform. Contrary to our hypotheses, mixed analysis of variance (ANOVAs) and T-tests showed more out-of-phase and variable hip-lower lumbar coordination compared to healthy controls, particularly at the start and end of the task. This suggested that presence of LBP was not sufficient to prompt a guarded response, although the task may not have been threatening/painful enough to elicit a response.

Chapter 4 identified a methodological issue with the Hilbert transform approach for CRP analyses (i.e., end-effects), which can bias CRP data and complicate the clinical interpretation of coordination and variability data. Thus, we investigated the impact of data padding techniques (double reflection, mirroring, spline extrapolation) to manage end-effects following Hilbert-transformed CRP calculations (Chapter 5). This was performed using lumbo-pelvic kinematic data from adults with LBP who were performing a sit-to-stand-to-sit task (n = 16). Root mean squared deviation (RMSD) and true error compared signals with a gold standard (extraneous data), and bootstrapping procedures tested for statistical significance. Results showed that spline extrapolation procedures best control for data distortion when analysing clinical data.

Lastly, we examined the relationship between pain-related threat and coordination in adults with chronic LBP (n = 49) during a lifting task (Chapter 6). Kinematic data of the spine and hips were collected using an electromagnetic system, and CRP analyses using a Hilbert transform determined hip-spine coordination amplitude and variability. Multiple regression analyses revealed that greater pain catastrophizing, but not fear, was independently related to more in-phase hip-lower lumber coordination amplitude. Exploratory analyses revealed two subgroups consistent with “tight” and “loose” control phenotypes, with the “tight” group showing elevated catastrophizing and disability.

Abrégé

La douleur peut servir de mécanisme de protection, incitant à protéger une région qui est perçue comme étant blessée ou menacée. Chez les personnes souffrant de lombalgie, une diminution du mouvement de la colonne vertébrale, la coordination hanche-colonne vertébrale plus raide, et la variabilité réduite, reflètent des comportements moteurs de protection. Bien que ces comportements puissent causer une charge tissulaire anormale et compliquer la convalescence, ils sont observés de façon incohérente dans la littérature et sont mal compris. Ces incohérences pourraient concerner (i) les considérations liées aux tâches, (ii) les procédures méthodologiques biomécaniques et (iii) l'influence des variables biopsychosociales. Une meilleure compréhension de ces relations pourrait combler les lacunes dans les connaissances et aider à éclairer les stratégies de traitement ainsi que les recherches futures. Par conséquent, l'objectif principal de cette thèse était de comprendre les altérations des comportements moteurs chez les personnes atteintes de lombalgie en étudiant les déterminants du mouvement de la colonne vertébrale et de la coordination de la colonne vertébrale. Cet objectif général a été atteint au moyen de quatre manuscrits.

Tout d'abord, une revue systématique et une méta-analyse ont résumé la relation entre le comportement moteur (c.-à-d. l'amplitude du mouvement de la colonne vertébrale, l'activité des muscles du tronc, et la coordination) et la menace liée à la douleur (par le biais de la catastrophisation envers la douleur et de la peur liée à la douleur), chez les adultes atteints de lombalgie (chapitre 3). La recherche de la littérature a été faite dans quatre bases de données et les articles ont été examinés et évalués par des évaluateurs distincts. Vingt et une études ont été retenues pour cinq méta-analyses, qui ont été réalisées à l'aide du modèle à effets aléatoires. Un petit effet associant une plus grande menace liée à la douleur avec un comportement moteur

protégé (c.-à-d., diminution de l'amplitude du mouvement de la colonne vertébrale, et une plus grande activité du muscle du tronc) pendant les tâches basées sur la flexion a été observé, mais pas de façon constante pendant les autres mouvements. Le manque d'études disponibles n'a pas permis de faire l'étude de la relation entre la menace liée à la douleur et la coordination.

Ensuite, nous avons comparé la coordination et la variabilité de la hanche-colonne vertébrale, au cours d'une nouvelle tâche, chez les personnes avec (n = 16) et sans lombalgie (n = 21) (chapitre 4). Nous avons utilisé des données cinématiques lombo-pelviennes recueillies précédemment à partir d'une tâche de position assise-debout-assise, et avons effectué des analyses de phase relative continue (PRC) à l'aide d'une transformation de Hilbert. Contrairement à nos hypothèses, l'analyse mixte de la variance (ANOVAs) et les tests-T ont montré une coordination hanche-lombaire plus décalée et variable que les contrôles, en particulier au début et à la fin de la tâche. Cela suggère que la présence de lombalgie n'était pas suffisante pour déclencher une réponse de protection, bien que la tâche n'ait peut-être pas été assez menaçante/douloureuse pour susciter une réponse.

Le chapitre 4 a relevé un problème méthodologique lié à l'approche de transformation de Hilbert pour les analyses du PRC (c.-à-d. distorsion de données), qui peut biaiser les données du PRC et compliquer l'interprétation clinique de la coordination et de la variabilité. Ainsi, nous avons étudié l'impact des techniques de remplissage de données (double réflexion, miroir, extrapolation spline) pour gérer la distorsion de données suite aux calculs du PRC transformé par l'approche Hilbert (chapitre 5). Ceci a été effectué à l'aide de données cinématiques lombo-pelviennes provenant d'adultes atteints de lombalgie qui effectuaient une tâche de position assise-debout-assise (n = 16). L'écart quadratique moyen et l'erreur vraie ont comparé les signaux avec un étalon-or, et les procédures de « bootstrapping » ont été testées pour la signification statistique.

Les résultats ont montré que les procédures d'extrapolation spline contrôlent mieux la distorsion des données lors de l'analyse des données cliniques.

Enfin, nous avons examiné la relation entre la menace liée à la douleur et la coordination chez les adultes atteints de lombalgie chronique (n = 49) pendant une tâche de lever de charge (chapitre 6). Les données cinématiques de la colonne vertébrale et des hanches ont été recueillies à l'aide d'un système électromagnétique, et les analyses PRC à l'aide d'une transformation de Hilbert ont déterminé l'amplitude et la variabilité de la coordination hanche-colonne. Des analyses de régression ont révélé qu'une plus grande catastrophisation de la douleur, mais pas la peur liée à la douleur, était indépendamment liée à une amplitude de coordination plus en phase entre la hanche et la colonne vertébrale. Les analyses exploratoires ont révélé deux sous-groupes correspondant à des phénotypes « raides» et « relâchés », avec le groupe « raide » démontrant un taux élevé de catastrophisation et de l'invalidité.

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Contribution to original knowledge

This Ph.D. dissertation includes four original manuscripts which comprise chapters 3 through 6. Chapters 3 through 5 have been published in peer-reviewed journals. Chapter 6 in is preparation for submission to a peer-reviewed journal.

In chapter 3, the manuscript "*The relationship between pain-related threat and motor behavior in people with non-specific low back pain: a systematic review and meta-analysis*" summarized the relationship between motor behavior and pain-related threat. While under this manuscript was under review, a similar systematic review was published in a different journal. Our manuscript extended their findings, showing a small effect between pain-related threat and guarded motor behavior during flexion-based tasks. Further, we provided an in-depth discussion of the clinical implications of these findings for rehabilitation professionals.

In chapter 4, the manuscript "*Movement variability in adults with low back pain during sit-to-stand-to-sit*" was the first manuscript to compare coordination amplitude and variability during a sit-to-stand-to-sit task in adults with and without LBP, using continuous relative phase analyses. Further, it was the first to explore coordination from the perspective of hip-lower lumbar and lower lumbar-upper lumbar joint pairs, while partitioning the sit-to-stand task into distinct periods (i.e., standing up vs. sitting down).

In chapter 5, the manuscript "*The effects of data padding techniques on continuous relative phase analysis using the Hilbert transform*" addressed a methodological issue associated with biomechanical procedures used to calculate coordination and variability that can introduce measurement error. We drew on past literature and tested data padding procedures to help control this issue. We detailed a novel solution using both theoretical and clinical data, which will help

inform future researchers studying coordination and variability using a Hilbert transform for continuous relative phase analyses.

Lastly, in chapter 6, the manuscript titled "*The relationship between pain-related threat and coordination in adults with chronic low back pain during a lifting task: a cross-sectional study*" was the first to study the relationship between threat and coordination using a more threatening task. We were the first to connect greater pain-related threat and guarded motor behaviors during lifting, from the perspective of coordination and variability in the hip-lower lumbar spine.

Contribution of authors

Mr. Patrick Ippersiel was the first author on all four manuscripts in this Ph.D. thesis because he led all aspects of this dissertation including: developing the research questions and research design; obtaining research funding; participant recruitment and data collection (chapter 6); data processing, analyses, and interpretation; manuscript preparation; and knowledge translation activities. Given chapters 4 and 5 used an existing dataset from Dr. Richard Preuss, Patrick Ippersiel was not involved in participant recruitment or data collection for these studies.

For chapter 3, Anthony Teoli is listed as second author in recognition of his contribution to the data extraction process and study quality appraisal. Dr. Shawn Robbins provided methodological expertise, advice regarding data analyses, and served as a third reviewer during the extraction process. Dr. Shawn Robbins, Dr. Timothy Wideman, and Dr. Richard Preuss all provided feedback and insight during writing of this chapter. Mrs. Jill Boruff helped develop a search strategy.

Chapters 4 and 5 utilized an existing dataset belonging to Dr. Richard Preuss. Dr. Preuss was responsible for participant recruitment and data collection. Otherwise, Dr. Shawn Robbins and Dr. Richard Preuss contributed to research design, data analysis, and provided feedback and insight during the writing process.

Chapter 6 analysed a new dataset. All data collection and participant recruitment were performed by Patrick Ippersiel. Dr. Shawn Robbins, Dr. Richard Preuss, and Dr. Timothy Wideman contributed to research design. Dr. Ian Shrier provided advice on statistical analyses. Dr. Shawn Robbins provided ongoing assistance with data analysis and interpretation. All authors provided feedback and insight during the writing process.

Chapter 1: Introduction

1.1. Low back pain

1.1.1. The burden of low back pain

Low back pain (LBP) is the leading cause of disability in the world (1, 2). This condition is extremely common, as approximately 85% of people experience an episode in their lifetime (1). While some episodes are self-limiting, many develop persistent disabling symptoms, which can negatively impact multiple aspect of their lives (3). In 2015, LBP was responsible for close to 60 million years lived with disability, a figure which represents a 50% increase in the past 25 years (2). The net result is a massive socio-economic burden, with estimated annual indirect costs ranging from $18.5 to $28.2 billion, in the United States alone (4). Despite decades of research and health-care expenditures, the burden of LBP to continues to increase (2, 5). As a result, LBP remains of significant societal concern.

1.1.2. What is low back pain?

Low back pain refers to pain that is primarily located between the lower ribs and gluteal folds (6). Low back pain can be categorized as: (i) specific low back pain, (ii) low back pain with neurological symptoms, and (iii) non-specific low back pain (7). Specific LBP reflects more serious spinal pain of a known origin (e.g., cauda equina syndrome, malignancy, infection, axial spondyloarthritis) (7). Specific LBP is quite rare (< 1% of all LBP (8)) and may require urgent or targeted medical management. Low back pain with neurological symptoms is more common (< 10% of all LBP) and can be described as radicular pain and/or radiculopathy (previously termed sciatica) (7). This class of LBP is characterized by leg-dominant pain with or without neurological compromise of a nerve root, often overlapping with pathologies such as disc prolapses, high-grade spondylolistheses, and/or central or foraminal spinal stenosis (9, 10).

Approximately 90% of LBP is regarded as non-specific, meaning no specific nociceptive source (i.e., tissue damage) can be identified as the cause of back pain (7, 10). Non-specific LBP may occur with or without leg pain; however, the leg pain is neither the dominant symptom, nor is it radicular in nature (11). The diagnosis of non-specific LBP is made via a triage process involving history taking and clinical examination, thereby ruling out any serious pathology (10, 12). Non-specific LBP can be described in terms of the length of time that symptoms persist. Specifically, LBP can be acute (symptoms lasting less than 6 weeks), sub-acute (symptoms lasting 6-12 weeks), or chronic (symptoms lasting longer than 12 weeks). Both specific LBP and LBP with neurological symptoms are unique classes of LBP, differing in presentation and management from non-specific LBP (7). For these reasons, specific LBP and LBP with neurological symptoms will not be further discussed in this thesis. For clarity, unless stated otherwise, all future references to LBP will be in relation to non-specific LBP.

1.1.3. Physical and psychosocial risk factors for low back pain onset

Risk factors for sudden onset LBP are broadly linked to physical, psychosocial, genetic, and lifestyle factors; however, causal mechanisms remain unclear (7). A recent systematic review reported prior history of back pain as the most consistent risk factor for future LBP, while no other metrics were consistently identified (13). A separate review reported that acute LBP may be precipitated by physical factors (i.e., exposure to manual tasks), but highlighted the cumulative effect of cognitive distraction and a physical task as increasing risk for future back pain (14). Otherwise, an individual's genetic make-up (15); the presence of co-morbid chronic or mental health conditions (e.g., diabetes, depression) (15, 16); and lifestyle factors associated with poor health such as obesity (17) or low levels of physical activity (18) are all linked to the occurrence

and/or persistence of LBP. Together, this evidence underscores the importance considering physical factors alongside other biopsychosocial factors when conceptualizing risk for LBP.

1.1.4. Course of low back pain and re-occurrence rate

While LBP is often regarded as self-limiting and a favourable outcome is expected in many, a subset of individuals is prone to developing persistent disabling symptoms (7, 11). A meta-analysis (13 cohorts, 11 113 participants) examining trajectories in people with LBP found that most people with acute LBP (< 6 weeks in duration) are substantially improved at 6 weeks and, on average, have low pain levels at 1-year (6/100 pain scale, 95% Confidence Interval (CI) = [3-10]). In contrast, people with LBP that persists longer than 6 weeks (i.e., sub-acute +/- chronic) could expect to have moderate levels of pain and disability at 1-year (23/100 pain scale, 95% CI = [16-30]) (19). Other work estimated that 65% of people with LBP (defined as < 3 months in duration) still reported symptoms at 3 and 12 months post onset (20). Relapses in LBP are also common. A high-quality inception cohort study (250 participants) reported that 69% of participants had a recurrence of a LBP episode within 1 year, of which 40% had to limit their activities, and 41% seeked care (21). In sum, this evidence challenges the notion of widespread spontaneous recovery in LBP and suggests that many people continue to experience pain and disability after an initial episode.

1.2. The biopsychosocial model for low back pain

1.2.1. The biomedical model for low back pain and its shortcomings

Traditionally, LBP has been conceptualized within a biomedical model. A biomedical model presumes that pathologies such as intervertebral disc herniations, facet joint degeneration,

spondylolisthesis, spinal stenosis, or vertebral endplate (i.e., Modic) changes are the primary drivers of low back pain and disability (22). While this is accurate for some (23), the biomedical model is challenged by the presence of "pathological findings" on MRI in asymptomatic individuals; by poor correlation of these "pathological findings" to pain and disability in people with LBP (24-27); and by the inability of clinical tests to reliably characterize spinal structures as painful (28). Further, the biomedical model overlooks a significant body of literature which suggests that a complex interplay of psychological, social, neurophysiological, and physical factors, help drive pain and disability in LBP (26, 29). Thus, in most individuals with LBP (i.e., non-specific LBP), pathology and tissue damage alone are not sufficient to explain the presentation and persistence of their symptoms.

1.2.2. The biopsychosocial model for low back pain

Over 40 years ago, Engel proposed an expansion of the biomedical model to include the consideration of psychological and social determinants of health, alongside biological factors (30). This was termed the biopsychosocial model. In 1987, Gordon Waddell's seminal manuscript applied this theoretical framework to assess and treat LBP, and advocated for a shift towards a whole-person perspective when managing LBP and LBP-related disability (31). This model was quickly popularized and has since become an important framework for assessing and managing chronic pain conditions, including LBP (29, 32). The biopsychosocial model is grounded in the view that a complex interplay of biological (or biophysical), psychological, and social factors impact pain and disability in people with LBP. The relative importance of presenting factors (e.g., nociception, pain-related fear) is unique to the individual and their experience, and it is critical to retain that there are no firm boundaries between domains. The following sections will provide an

overview of the different domains of the biopsychosocial model and discuss how they relate to LBP and LBP-related disability.

1.2.3. Biological (biophysical factors)

Biological, or biophysical factors relate to the underlying biology and mechanics of the musculoskeletal and nervous systems, encompassing patho-anatomical, physical, and neurobiological considerations (33). First, patho-anatomical factors reflect tissue pathology (e.g., disc herniation) as a possible driver of pain (23, 24). Physical factors can be characterized by exposure to mechanical spinal loading during functional activities. In LBP, repeated exposure to heavy lifting, bending, and other daily activities have been linked in varying degrees to disabling LBP (34, 35). Physical factors also encompass an individual's underlying motor behavior (i.e., movement biomechanics). It is common for people with LBP to move slower (36), have less spinal range of motion (36), and to show greater levels of muscle activity (37) during functional tasks, when compared to healthy individuals. These behaviors are proposed to increase spinal/tissue loading and impact and pain and disability in the long-term (38-40). Finally, physical factors can also be considered in terms of alterations in trunk muscle morphology and composition (41, 42); however, the clinical implications of these findings are less clear.

Neurobiological factors are best understood when considering that pain is more complex than processing of nociceptive input from the periphery related to tissue damage. Rather, nociceptive and sensory input can be modulated by a wide range of factors at the periphery, the spinal cord, and at supraspinal regions; all of which may or may not contribute to a painful response in an individual (43, 44). This modulation can be linked to neurobiological (e.g., structural/plastic) changes in the central nervous system, which can create an environment of hypersensitivity to

stimuli and pain. This phenomenon is known as central sensitization and is a common feature of many chronic pain conditions, including LBP (43, 44).

1.2.4. Psychological factors

Psychological factors can be broadly regarded as thoughts, feelings, and related behaviors which may negatively influence clinical outcomes in people with LBP. Within this broad conceptualization, several overlapping constructs have emerged as important predictors of negative pain-related outcomes in people with disabling LBP, namely, pain catastrophizing (45, 46), pain-related fear (47-49), self-efficacy (50), and depression (51). Of these, pain catastrophizing and pain-related fear are commonly studied constructs, in-part due to their important implications in clinical rehabilitation (32).

Pain catastrophizing reflects a negative outlook on pain and is characterized by rumination, magnification of the threat value of pain, and a sense of helplessness (45, 46). In contrast, pain-related fear is defined as fear that emerges when stimuli related to pain are perceived as threatening (48). Both constructs are important predictors of pain and disability in LBP and are core elements of popular models used to explain pain and disability (i.e., Fear-avoidance model) (47-49). For example, a meta-analysis involving 5510 participants over 20 studies found a robust positive association between pain-related fear and LBP-related disability, that was moderate to large in size ($N = 5510$, $k = 20$, $r^- = 0.42$) (52). In parallel, a systematic review of 11 randomized controlled trials studying the effect of catastrophizing in LBP reported baseline catastrophizing scores to be predictive of treatment outcomes in LBP (four studies), with higher catastrophizing scores being associated with greater disability at follow-up (four studies) and greater pain severity (two studies) (53).

1.2.5. Social, lifestyle, and other factors

There are many other important factors when considering disabling LBP from a whole person perspective. For example, low education, low socio-economic status, and unemployment are all associated with increased chronic LBP prevalence (54). Lifestyle factors such as increased BMI, smoking, poor sleep, and levels of physical activity are also linked to LBP (26). Additionally, there is also evidence that an individual's genetic predisposition may increase the risk of developing chronic LBP (55).

1.3. What is being done for low back pain?

Due to the lack of an identifiable source of pain in non-specific LBP, the management of this condition is aimed at reducing pain and disability (10). For acute LBP, clinical practice guidelines recommend education and advice to stay active, analgesic medicines, select non-pharmacological therapies (e.g., spinal manipulation), and timely review based on patient presentation (10, 56). For chronic LBP, guidelines are fairly aligned with those for acute LBP; however, they prioritize exercise, and recommend managing comorbidities (e.g., depression), monitoring prolonged analgesic use, and using psychologically informed therapies (e.g., cognitive behavioral therapy) (10, 56).

From the perspective of rehabilitation professionals, exercise therapy is a core component of treatment programs and is recommended as a first-line intervention for chronic LBP (10, 56). There is high quality evidence of its effectiveness in chronic LBP compared to sham or placebo interventions (mean difference in pain ratings /100 = −8.58, 95% CI = [−18.46 to −1.29]); however, the effects are rather modest and similar to those of other conservative interventions (10, 57). Part

of this small effect may relate to the fact that greater pain during exercise is often cited as a barrier to treatment adherence, a factor which may negatively influence treatment outcomes (58). While it is unclear as to why an increase in pain during exercise occurs in some, one explanation could relate to altered motor behavior (i.e., movement quality) that is consistently observed in people with chronic LBP (36-39). These altered behaviors are thought to sub-optimally load tissues, a process which may exacerbate pain during exercise programs and help fuel a cycle of persistent symptoms, re-injury, and disability (38, 39). Through this lens, improving movement quality could help improve treatment adherence and clinical outcomes.

1.3.1. Motor behavior, clinical rehabilitation, and general research gaps

In the spine, motor behavior is an overarching term used to describe the underlying biomechanics of movement and can be quantified through measures of spinal range of motion (ROM), trunk coordination and variability, and muscle activity. This area of research has formed an exciting space for the past 30 years; however, many gaps in our understanding of this construct remain. Traditionally, researchers conceptualized motor behavior from the perspective of ROM and trunk muscle activity (36, 37). These constructs have quantified how much movement or muscle activity is occurring in an individual with LBP; however, they have had limited value in improving clinical outcomes (59). One emerging area relates to the study of joint coordination and variability. The study of coordination and variability in LBP diverges from traditional metrics (e.g., spine ROM) in that it describes *how* an individual in pain moves, rather than simply *how much* one moves. For instance, coordination describes relationships between joints (e.g., hip-spine coordination) across an entire movement cycle, whereas variability points to how this pattern of coordination changes across multiple repetitions of a task. By extension, this provides insight into motor control and the underlying mechanics of how movement is organized. Clinically, better

understanding of coordination and variability may provide further opportunity to modify movement patterns to help rehabilitate an individual in pain (60). From this perspective, changing movement could modify loading on the spine and help improve outcomes in people with LBP (61). Currently, research in this area is lacking, and the existing literature is conflicting from both a clinical and methodological standpoint (62).

One major limitation of biomechanical research in LBP is that motor behavior is often considered as a stand-alone construct. While there is general consensus that motor behavior plays a role in the development/maintenance of LBP, one cannot discount the potential influence of biopsychosocial factors on this construct. With the emergence of biopsychosocial models of care for LBP, it is increasingly apparent that motor behaviors (e.g., coordination and variability) are but one clinical consideration alongside other important constructs such as psychological risk factors (e.g., pain-related fear).

Therefore, the overarching goal of this thesis is to understand motor behaviors in chronic LBP, from the perspective of spinal motion and joint coordination and variability. This will be discussed while considering both traditional (i.e., methodological, clinical) and biopsychosocial (i.e., pain-related fear) factors. The starting point in this discussion relates to understanding how pain changes movement and will be reviewed chapter 2.

1.4. Chapter 1 references

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Chapter 2: Background

2.1. Pain and movement: a complex relationship

2.1.1. Traditional theories

It is generally accepted that people in pain move differently than healthy individuals (1-3). The underlying mechanism(s) in this process, however, are poorly understood. Traditionally, two theories have attempted to explain this phenomenon: the vicious cycle theory (4), and the pain adaptation theory (5). The vicious cycle theory proposes that pain results in a prolonged increase in muscle activity via spinal mechanisms (i.e., spasm). Spasms were thought to result in ischemia and the accumulation of metabolites, a process which would then produce more pain and muscle activity, resulting in a vicious cycle (pain-spasm-pain cycle) (4). In contrast, the Pain Adaptation Theory proposed that pain leads to stereotypical inhibition of painful agonist muscles and facilitation of antagonist muscles. This process was thought to be mediated by spinal and brainstem mechanisms, and reflect an attempt to reduce movement of the injured area (5). Both models, however, predicted a stereotypical motor response to pain and were unable to explain the vast heterogeneity of within- and between-individual variability in motor responses of people in pain (1, 6). Further, they were not aligned with a biopsychosocial understanding of pain and could not account for "top down" mechanisms capable of influencing motor behavior (e.g., negative cognitions) in the absence of peripheral nociceptive input (7).

2.1.2. The motor adaptation to pain theory

Hodges and Tucker's seminal manuscript extended the works of their predecessors, and developed a new motor adaptation to pain theory (1). This theory proposed that in response to pain, injury, or threat of pain/injury, the nervous system prompts a short-term motor response aimed at protecting injured or threatened structures (e.g., guarding/splinting). Analogous to limping

following an acute ankle sprain, this change in motor behavior is thought to be protective/functional and encourage tissue healing. The motor adaptation to pain theory diverges from past models in that the predicted motor response to pain is highly individualized, occurs over a spectrum of motor behaviors, and is influenced by a range of personal (e.g., task, context) and biopsychosocial factors. As a result, it can explain motor responses which range from subtle changes in motor behavior due to pain (e.g., minor re-organization of motor units (8)) to complete movement avoidance (9). Further, this theory also recognizes the potential influence of both bottom up (e.g., peripheral nociceptive input (10)) and top-down (e.g., cognitive and emotional) mechanisms (11). Overall, the motor adaptation to pain theory provides a comprehensive framework to better understand the heterogeneity in motor responses observed in people with LBP, embedded in a biopsychosocial understanding of pain.

2.1.3. Short term protection but long-term consequences in low back pain?

Hodges and Tucker's theory asserts that the underlying goal of all motor responses to pain (or threat thereof) is that of protection (1, 6). While this protective response makes sense with acute LBP, the need for protection in people with chronic LBP is less clear, particularly when LBP is often non-specific and has surpassed the timeline of expected tissue healing (1, 6, 12). In LBP, protective motor behaviors are inferred using biomechanical analysis and are linked to observations of: (i) increased superficial trunk muscle activity (13-15); (ii) decreases in trunk mobility and movement speed (2); and (iii) a loss in variability of motor strategies (16, 17). Together, these behaviors are thought to load spinal tissues and structures sub-optimally and may act as an ongoing source of nociception, helping perpetuate pain and disability in chronic LBP (18-

20). Due to their important links with exercise and clinical rehabilitation, these observations of altered motor behavior in people LBP will be discussed in greater detail.

2.1.3.1. Increased trunk muscle activity

Greater trunk muscle activity is likened to guarding/bracing and is a common characteristic in people with LBP. A meta-analysis reported elevated trunk extensor muscle activity in people with LBP, compared to healthy adults, during bending (Standardized Mean Difference (SMD)= -1.71, 95% CI = [-2.25 to -1.35]) and standing (SMD = 1.14, 95% CI [-0.03 to 4.81]), but not during other tasks (e.g., sitting, rotation) (13). Others reported greater extensor and/or superficial abdominal muscle activity in people with chronic LBP during sitting (21), walking (22-25), tasks involving trunk perturbations (26), and during a battery of functional tasks (14). These observations, however, need to be considered alongside fairly consistent evidence of inhibition/delays of deep trunk muscles (i.e., multifidus and transverse abdominis) in people with LBP (27). Thus, while there is evidence that, on average, motor behavior in chronic LBP is changed towards a net increase in superficial trunk muscle activity, this may be a compensatory strategy to account for inhibition of deep trunk musculature (i.e., decreased spinal control).

Excessive trunk muscle activity during movement is understood to increase trunk stiffness and compressive forces on the spine (18, 19, 28). While a degree of load is likely beneficial for tissue regeneration and building capacity (29), excessive and sustained load may accelerate spinal degeneration and hamper tissue healing (30, 31). For instance, animal model studies linked sustained compressive load to increased disc degeneration, possibly due to disrupted fluid mechanics of the disc (32). Alternatively, sustained compressive loading may act as a source of nociceptive input on load intolerant structures in the spine (1, 27). In sum, the role of greater trunk

muscle activity as a long-term protective strategy is debateable and likely limits motion at the spine, contributing to other potentially problematic behaviors observed in LBP (i.e., reduced ROM and reduced variability)

2.1.3.2. Decreased spinal range of motion

Reduced spinal range of motion (ROM) during functional movement is common in people with LBP and likely reflects a response to pain (33), and/or a compromise of structural tissue (34). A meta-analysis studying spinal kinematics in people with and without LBP, provided robust evidence that people with LBP generally have reduced ROM compared to healthy individuals during spinal flexion (SMD = -0.62, 95% CI = [-0.94 to -0.29]), spinal extension (SMD = -0.54, 95% CI = [-0.81 to -0.27]), lateral flexion (SMD = -0.73, 95% CI = [-1.14 to -0.33]), and rotation (SMD = -0.49, 95% CI = [-0.76 to -0.22]) (2). This review also reported reduced speed/acceleration of movement (SMD = -1.24, 95% CI = [-1.58 to -0.90]) in people with LBP, during a variety of tasks (2). Decreased ROM and movement speed are thought to impair shock absorption and dampening of forces exerted on the spine, further increasing loading during movement (20, 26). However, in contrast with the above-mentioned studies linking trunk muscle compressive loading with possible sources of nociception, to the author's knowledge, there is sparse literature studying the impact of decreased spinal motion on spine and tissue mechanics. Therefore, this latter relationship remains more theoretical at this point.

2.1.3.3. Movement variability

There is sufficient evidence to suggest that, on average, people with LBP move with greater levels of trunk muscle activity and reduced spinal ROM, compared to healthy individuals (2, 13).

The role of movement variability (i.e., how variable an individual's movement strategy is across multiple repetitions of a task) in LBP, however, is more complicated and nuanced from both a clinical and methodological standpoint. For instance, a recent scoping review on movement variability and LBP identified 7 distinct methodologies to measure variability, each with their own strengths, weaknesses, and unique clinical implications (35). Further, these methodological differences may be in-part driving inconsistent observations of movement variability in people with LBP. Movement variability forms a central tenet of this thesis, and to better understand this construct, theoretical, methodological, and clinical considerations for movement variability will be reviewed in detail.

2.2. Movement variability and the Bernstein perspective

2.2.1. Movement variability: a theoretical overview

Variability is a fundamental feature of human movement (36). This conduct is defined as the normal variations that occur in motor performance across multiple repetitions of a task, and is understood to reflect the nervous system's flexibility in how muscles and joints are coordinated during movement (37, 38). The study of variability has long been tied to the "degrees of freedom problem", wherein researchers have attempted to understand redundancy in the motor system. The underlying question with motor redundancy relates to how the central nervous system chooses to coordinate movement, when there are many motor options available to complete a task (39).

Nikolai Bernstein's classic experiment on blacksmiths striking a chisel with a hammer provided insight into this question (40). Bernstein observed that high within-individual variability in how the upper extremity was coordinated/organized (in terms of joint angles) across multiple trials was linked to low variability of the hammer trajectory (i.e., consistently striking the chisel).

This was described as “repetition without repetition”, that is, repetitive completion of a task via many possible means (39, 40). Given the blacksmiths were considered experts at striking a chisel, the observation of greater within-individual variability in how this task was completed challenged the notion of the existence of an ‘optimal’ movement pattern. Bernstein’s findings were later replicated in animals and humans, challenging the traditional perspective that variability is the result of error and linked to poor motor performance (39, 41, 42). Together, this provided evidence that variability is functional and that: (i) individual parts (e.g., joints/muscles) can be organized in many ways to serve a coordination pattern; and (ii) variability can provide insight into how these coordinative patterns are maintained across multiple repetitions of a task (43).

Broadly, there are two forms of movement variability: end-point variability (i.e., striking the chisel with a hammer) and coordinative variability (i.e., how this task was performed across multiple iterations) (43, 44). These distinct forms of variability have different interpretations. End-point variability is the traditional focus of biomechanics research and studies the outcome of a task, where low variability is likened to skilled performance of said task (43). In contrast, coordinative variability reflects how movement is organized to complete a task, and greater variability (within the limits of the physical system) is suggestive of robust and adaptable movement (43). From this perspective, the study of coordinative variability provides insight into motor control and might have key implications for musculoskeletal injury (discussed below) (38, 43). Therefore, the view put forth in this thesis is aligned with the Bernstein perspective, in that, coordinative variability is functional and essential for movement and performance (38-40, 43, 44).

Scholz, Schoner, and Latash developed the uncontrolled manifold hypothesis to explain how the nervous system controls movement variability to achieve a task-specific goal (45, 46). Essentially, the theory proposes that the CNS utilizes available degrees of freedom to ensure stable

and flexible performance of motor tasks. This is easily understood in the context of an example; thus, let us consider an upper extremity pointing task where the goal is to touch one's nose with the index finger. The uncontrolled manifold hypothesis states that the controller first defines a common "joint space" in which all analysis takes place; this space may contain all joint angles involved in the task (e.g., 7 major rotations shared by the shoulder, elbow and wrist). Within that space, the controller organizes a subset (the manifold), which would contain all sets of joint angles that correspond to a specific performance variable associated with a successful task (e.g., Cartesian coordinates of fingertip position touching the nose). The nervous system then organizes these elemental variables (e.g., joint rotations) in such a way that limits most of the variability to within the subset (manifold). Thus, we would expect considerable variability in elemental variables, while maintaining a stable performance variable (i.e., there are countless combinations of joint configurations associated with successfully touching your nose). The general premise is that the controller exerts minimal control over the elemental variables if they stay within the manifold, hence the term "uncontrolled". The uncontrolled manifold hypothesis is primarily concerned with structure of variability within the manifold and can be categorized as "good" or "bad". Good variability is associated with system flexibility and no task violation, while variability which introduces performance error is "bad". In other words, the CNS will preferentially stabilize sets of joint angles relevant for stabilization of the performance variable over sets of joint angles that do not.

Due to some ambiguity surrounding the term movement variability in the literature, the following section will review key methodological and conceptual distinctions when discussing movement variability. This will be followed by an in-depth literature review of the constructs of **coordination amplitude** and **coordinative variability** from: (i) methodological, (ii) clinical (e.g.,

task considerations, comparing people with and without LBP), and (iii) biopsychosocial (i.e., relationships with pain-related fear and catastrophizing) standpoints.

2.3. Movement variability: conceptual and methodological clarifications

2.3.1. Movement variability: methodological considerations

In biomechanical research, due to differences in availability of laboratory equipment/software, technical expertise, and constant evolution of the literature base, similar research questions may be answered using distinct methodological approaches. In turn, differences in methodology between studies may lead to different interpretations of findings and complicate overall synthesis of the evidence base. For instance, a recent scoping review identified 7 different methodologies used to quantify movement variability of the spine in people with LBP (35). Traditionally, variability was simply quantified from the standpoint of magnitude and quality of variation (47). This involved using metrics such as the range or standard deviation (SD) to reduce information across multiple trials (e.g., joint angles during gait) and were often summarized as the mean curve +/- its SD (47, 48). The main drawback of these approaches is that they do not provide information about the time-evolving nature of the signal, nor do they provide robust insight to the underlying dynamics of movement (47). As a result, they may be of limited clinical relevance.

The other subset of tools used to quantify variability are those based in Dynamics Systems Theory (49) (e.g., Lyapunov exponent, Continuous Relative Phase). In brief, Dynamic Systems Theory proposes that biological systems self-organize based on biomechanical (i.e., Newtonian laws), environmental (e.g., spatial and temporal configuration of events), task (e.g., walking fast or slow), and morphological constraints (i.e., biological constructs), to find the most stable solution

when producing voluntary movement (36, 47, 50). Thus, Dynamic Systems Theory serves as a framework to study how different elements (e.g., joint segments, environment/task constraints, morphological factors) interact in a time-evolving nature. Within this framework, researchers have opted to study variability from the perspective of: (i) coordinative variability, that is, how two adjacent joints/segments interact during cyclic movement (e.g., hip-spine coordination during repeated lifting); and (ii) in terms of stability and complexity of these coordinative patterns across multiple trials (44). While concepts of coordinative variability and stability/complexity are related, they remain distinct constructs and generate unique information on motor control and musculoskeletal injury (36, 44). In the literature, these terms are often used interchangeably and tend to get categorized under the umbrella term of movement variability – a practice which complicates the interpretation of an already complex body of literature. Therefore, for purposes of clarity, this thesis will view movement variability from the standpoint of the constructs of **coordinative amplitude** and **coordinative variability** as described by Hamill et al., and discuss methodologies and review the literature through this lens (43).

2.4. Coordination amplitude and variability: measurement

2.4.1. Measuring coordination amplitude and coordinative variability

The study of coordination amplitude and variability begins with an understanding of coordination. In the field of biomechanics, coordination can refer to how two adjacent joints/segments are moving relative to one another. Coordination amplitude, in comparison, simply reflects the extent to which this relationship is occurring (e.g., how in-phase or out-of-phase two joints/segment behave during movement). Joint coupling is a common term used to describe this behavior, where coupling implies that the motion in one joint can influence the motion of another

related joint (e.g., knee-hip coupling during gait) (43). In contrast, coordinative variability reflects the extent to which one's coordination strategy changes during repetitive movement (e.g., knee-hip coupling across consecutive gait cycles) (48). In this context, coordinative variability is simply a measure of how variable an individual's coordination amplitude is. Broadly, there are three approaches to quantifying coordination amplitude and coordinative variability: (i) discrete relative phase, (ii) continuous relative phase, and (iii) vector coding (43, 51, 52).

2.4.1.1. Discrete relative phase methods

Both discrete relative phase and continuous relative phase approaches consider phase relationships between two oscillating joints/segments. In turn, this provides insight into how two joints are moving, relative to one another. Discrete relative phase analysis, specifically, describes a temporal relationship in joint coupling. This approach evaluates the timing of key events during movement (e.g., maximal angular position), using the time-series of two joint/segment angles (43, 51). The resulting value occurs on a continuum, reflecting events occurring at the same instant (i.e., in-phase), or with a lag (i.e., out-of-phase) (43, 51). The main drawback of this approach is that usually only one data point of the time signal is used for analyses. Under these conditions, a regular, repeated movement cycle is often required (53). Unfortunately, this may be a challenge when studying human movement.

2.4.1.2. Continuous relative phase: phase plot approach

Continuous relative phase (CRP) analyses describe coordination between two joint angles/segments across an entire movement cycle (52, 54, 55). In this approach, joint/segment velocity and position data are used to construct separate phase plots for two joints/segments (e.g.,

hip and knee joints). Using these plots, instantaneous phase angles can be determined by calculating the four-quadrant arctangent, relative to the horizontal axis, for each joint segment/angle (51, 52). The CRP amplitude of the joint couple (e.g., hip-knee) is determined by subtracting the phase angle of the distal joint/segment, from that of the proximal joint/segment (55). The net result is a CRP amplitude curve for a joint couple (e.g., knee-hip) corresponding to one movement cycle, which is reflective of **coordination amplitude** (i.e., how in-sync two joints are moving relative to one another). Importantly, measures of **coordinative variability** are quantified by calculating the extent to which coordination amplitude varies over multiple trials of a task, within an individual. While this approach has been applied to study LBP and musculoskeletal injury (1, 16, 17, 52, 56), there are concerns with normalization procedures introducing artifacts when data is non-sinusoidal, as is often the case with kinematic data (55, 57). Recent recommendations suggest a third approach involving a Hilbert transform (55).

2.4.1.3. Continuous relative phase: Hilbert transform approach

The main difference with this approach is that phase angles are derived using a Hilbert transform, rather than a position–velocity plot. Briefly, a Hilbert transform converts a non-sinusoidal time-varying kinematic waveform into an analytical signal. Using the kinematic signal (e.g., hip joint angles) and its respective Hilbert transform at each point in time, one can calculate the phase angle for a joint/segment. This approach bypasses the need for signal normalization, helps mitigate data distortion, and produces a more robust estimate of CRP values (55). While resulting **coordination amplitude** values may be manipulated to [-180°, 180°] or [0°, 360°] ranges, data are commonly expressed on a [0° to 180°] scale. Here, 0° indicates fully in-phase relationships between two joints/segments (i.e., fully coupled) meaning the joints/segments are

moving in the same direction at the same time. A value of 180° indicates fully out-of-phase behavior (i.e., uncoupled), suggestive that joints/segments are moving in opposite directions, at the same time (43). As described above, measures of **coordinative variability** indicate how coordination amplitude varies during repetitive movement, where lower values reflect less variable coordinative patterns, and vice versa. Coordination amplitude is often summarized using ensemble averaging and is described as the Mean Absolute Relative Phase (MARP) (58), while coordinative variability is quantified by a measure of standard deviation of the CRP (or coordination amplitude) curves, and is described as the Deviation Phase (DP) (Figure 2.1) (58). Full methodological details are provided in chapter 3.

2.4.1.4. Vector coding approaches

Vector coding procedures quantify relative motion between two joints or segments (i.e., coordination) using angle-angle plots of a time-normalized movement cycle. Vector coding defines a coupling angle based on the orientation of a vector connecting two consecutive time points, relative to the right horizontal (43, 59, 60). Angles are output on a [0, 360°] scale and specific ranges of values are defined using "binning procedures" to quantify (i) in-phase coordination, (ii) rotation of the distal joint/segment, (iii) out-of-phase coordination, and (iv) rotation of the proximal joint/segment. This approach has also been used to study coordination and variability in people with LBP during a variety of tasks (61-63).

2.4.2. Comparing CRP and vector coding procedures

The main difference between vector coding and CRP analyses is that vector coding is based on positional signals, while CRP is derived either from: (i) position and velocity signals (i.e., phase

plot approach), or (ii) procedures involving a Hilbert-transform. While both vector coding and CRP approaches are acceptable when quantifying coordination amplitude and coordinative variability from a Dynamic Systems Theory perspective, comparing findings from studies using these distinct methods should be made with caution (64). For instance, Miller et al. compared vector coding and CRP phase plot approaches using theoretical and experimental kinematic gait data, showing that the CRP phase-plot approach produced a somewhat more conservative estimate of coordinative variability. This suggests that the CRP phase-plot approach may be less sensitive in detecting change compared to vector coding, a finding typically attributed to procedures involving phase plane scaling/normalization in CRP analyses (64, 65). However, this latter study did not compare vector coding to CRP procedures involving a Hilbert-transform. Recall, one benefit of the Hilbert-transform approach for CRP analyses is that it bypasses the need for signal normalization (55). To the author's knowledge, direct comparisons between vector coding and Hilbert-transformed CRP approaches do not exist in the literature. Overall, while it appears that the Hilbert-transform approach is the most suitable method for CRP analyses, there does not appear to be a clear superiority of one of technique (i.e., CRP analyses vs. vector coding) above the other for quantifying coordination amplitude and variability.

2.4.3. Summary and methodological gaps in measuring coordination amplitude and coordinative variability

One fundamental issue with the study of coordination amplitude and coordinative variability is dissociating "biomechanical noise" from true variability in the signal (43, 50). As outlined above, not only does the method chosen to quantify coordination amplitude and coordinative variability influence the extent of observed variability, but so do subsequent steps in

terms of data processing (e.g., normalization) (55, 57). From this standpoint, and based on the recommendations of Lamb and Stockl, it appears CRP analyses using the Hilbert transform approach is the most robust methodology and produces a conservative estimate of coordination amplitude and coordinative variability (55). Therefore, this approach was selected as the core methodology in this thesis to quantify coordination amplitude and variability.

Implementing the Hilbert transform, however, can result in end effects in a waveform (i.e., data distortion), similar to the difficulties encountered during digital filtering (66). Such distortions would preclude interpretation of CRP waveforms at the start and end of the movement and complicate common procedures which involve partitioning a task into separate phases (e.g., spinal flexion vs. re-extension)(56). While methods of data padding have been proposed to address this issue (67, 68), there has been no systematic investigation and comparison of techniques, leaving biomechanical researchers with no clear instruction on best-practice procedures. Improving CRP methodology will result in more accurate estimates and will serve to inform future research.

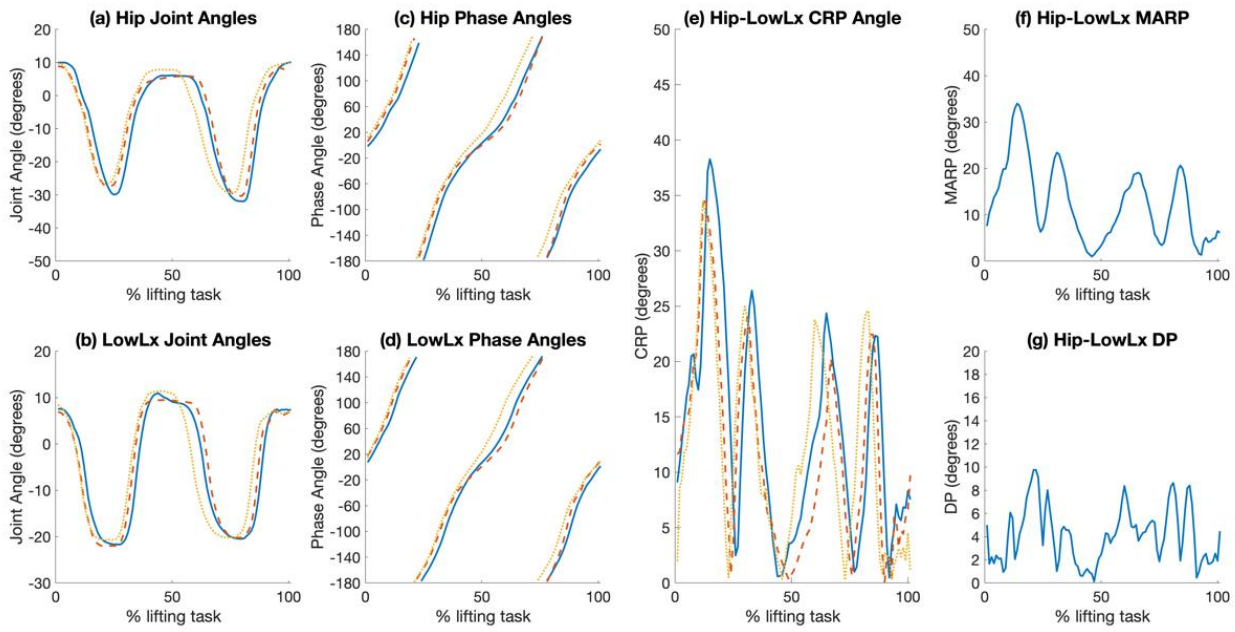

Figure 2.1. Methodological steps for continuous relative phase analyses: Measurement of Hip and lower lumbar (LowLx) joint kinematics during representative crate lift and replace trials (trial 1 solid blue line; trial 2 dashed red line; trial 3 dotted yellow line) and steps to determine continuous relative phase (CRP) angles. Graphs depict time normalized Hip and LowLx joint angles (a, b); Hip and LowLx phase angles determined using the Hilbert-transform approach (c, d); Hip-LowLx CRP angles on a 0-180 degree scale (e); Hip-LowLx Mean Absolute Relative Phase (MARP), which quantifies CRP amplitude across multiple trials (f); and Hip-LowLx Deviation Phase (DP), which quantifies CRP variability across multiple trials (g).

2.5. Coordination amplitude and variability, injury, and low back pain

2.5.1. Overview and implications for injury

Variability is a fundamental feature of human movement and speaks to how the nervous system coordinates the motor system to perform voluntary movement (36). From this standpoint, a healthy motor system is proposed to have an "optimal range" of variability (36). This variability renders the motor system adaptable and permits a rich repertoire of movement strategies when completing a task (37). Deviations from this "optimal" range of variability, however, are considered problematic and may be associated with altered tissue loading and musculoskeletal injury (Figure 2.2) (43).

This overarching concept forms an important starting point when discussing coordination, variability, and their implications for LBP. For purposes of clarity, the following section will further unpack this notion while reviewing the literature from the standpoint of coordination amplitude and coordinative variability. As a reminder, **coordination amplitude** allows the characterization of movement as more in-phase (e.g., tighter hip-spine coupling) or more out-of-phase (e.g., looser hip-spine coupling), while **coordinative variability** describes how variable these coupling strategies are, within an individual, during repetitive movement trials.

2.5.2. Literature review of coordination amplitude and coordinative variability in low back pain

Broadly, two presentations of coordination amplitude and variability in LBP are proposed to exist and are supported to some degree by the literature (27, 43). First, more in-phase lumbo-pelvic coordination amplitude and/or reduced coordinative variability are possible findings in people with LBP, with these observations being likened to protective/guarded motor behaviors.

This concept aligns nicely with observations of excessive trunk muscle activity (i.e., bracing), reduced spinal ROM, and the proposed phenotype of "tight" control in LBP (16, 27). While tight control may provide short-term benefit in terms of increasing spinal stiffness/stability following injury, persistence of these behaviors likely results in repeated compressive loading of spinal tissues and structures (43, 69).

In contrast, the opposite behavior of more out-of-phase coordination amplitude and/or excessive variability may be equally problematic in people with LBP. These behaviours are thought to reflect less robust control of the spine, where poor control is thought to predispose an individual to increased tissue loading and injury due to aberrant movement patterns (70). These behaviors are thought to be in-line with specific muscular inhibition and delays in responses to perturbations, tying in with a "loose" control phenotype in LBP (27) (Figure 2.2). There is evidence of both behaviours (i.e., in-phase and less variable vs. out-of-phase and more variable) in people with LBP, and the literature in these respective areas will be summarized.

2.5.2.1. Evidence for more in-phase coordination amplitude and decreased coordinative variability in low back pain (i.e., "tight" control)

The following section will review the LBP literature in which more in-phase coordination amplitude, reduced coordinative variability, or a combination thereof, have been reported. First, this has been researched from the standpoint of walking and running. There is robust evidence that people with LBP adopt a more in-phase trunk-pelvis coordination strategy during gait, compared to healthy individuals. A systematic review reported that 9/10 studies found this relationship (predominantly in the transverse plane) (71), with this behavior becoming more apparent during more complex tasks such as running (17, 63). Coordinative variability during gait and running has

been studied less. Seay et al. used CRP procedures and showed a robust loss of lumbo-pelvic transverse plane variability in people with chronic LBP during running ($P = 0.020$, effect size =1.11), but not walking ($P = 0.32$, effect size = 0.02) (17). Lamoth et al. used principal component analyses to describe less variable patterns of lumbo-pelvic transverse plane coordination in people with chronic LBP in response to sudden (72), and progressive, changes in velocity during treadmill walking (24). This more in-phase and less variable (i.e., "tight") behavior is likened to a "guarded gait" previously reported in people with LBP, although this is subject to debate in the literature (73, 74).

Coordinative amplitude and variability have also been studied from the standpoint of sagittal plane tasks such as bending and lifting. Mokhtarinia et al. used CRP procedures to study a flexion/extension task and compare the effects of movement asymmetry, velocity, and load, on lumbo-pelvic coordination amplitude and variability in people with and without chronic LBP. Their main findings were more in-phase (group main effect: $P = 0.005$) and less variable (symmetry x load x group interaction: $P = 0.03$) patterns of coordination in the LBP group (16). Importantly, differences in coordination amplitude were likely below the threshold of clinical significance (~2° between group difference) and changes in variability were only observed under more complex task conditions. Shojaei and colleagues used a prospective design comparing lumbo-pelvic coordination amplitude and variability, during fast and slow bending, in people with low-moderate (< 4/10 pain) and moderate-severe (≥ 4/10 pain) non-chronic LBP (< three months duration), and in healthy controls (75). At baseline, the low-moderate LBP group showed more in-phase (re-extension period, slow condition: $P = 0.018$) and less variable (bending period, slow condition: $P = 0.006$) coordinative behavior than healthy controls, and to a lesser extent, the moderate-severe group. At three months, comparisons between the low-moderate and moderate-

severe LBP groups showed that slow bending (low-moderate LBP ($\bar{x}$ and SD) = 2.57° +/- 1.15°, moderate-severe LBP: 4.76° +/- 1.72°; P = 0.039) and re-extension (low-moderate LBP: 1.78° +/- 1.15°, moderate-severe LBP: 4.70° +/- 1.72°; P = 0.001) were less variable in the low-moderate group, while coordination amplitude was indifferent (P > 0.147) (75). Interestingly, these behaviors persisted, despite improvements in pain intensity and disability, underscoring the complexity of altered movement behaviors in people chronic LBP. In a separate study on people with acute LBP, Shojaei et al. reported more in-phase coordination amplitude and less variable trunk-pelvis coordination during both forward bending (coordination amplitude: P = 0.025; coordinative variability: P = 0.002) and re-extension (coordination amplitude: P = 0.015; coordinative variability: P = 0.034), in comparison with healthy controls. Their findings suggest that protective behavior may be more present in the acute phase (76).

Overall, it appears there is some evidence of more in-phase and less variable (i.e., "tight") patterns of lumbo-pelvic coordination in people with LBP, compared to healthy controls. However, these findings were influenced by task complexity (e.g., adding load) and task period (e.g., bending vs. re-extension), which complicates synthesis of the findings. Additionally, these behaviors may be more apparent in acute LBP or in those with low-moderate LBP, but further research is required to flesh out this relationship.

2.5.2.2. Evidence for more out-of-phase coordination amplitude and increased coordinative variability in low back pain (i.e., "loose" control)

In contrast with the above-mentioned studies reporting more in-phase and less variable lumbo-pelvic coordination in people with LBP (i.e., "tight" control), there is evidence of the opposite behavior (i.e., "loose" control) in the literature. For instance, Silfies et al. used CRP

procedures to study lumbo-pelvic coordination amplitude and variability during a repeated reaching task with loaded and non-loaded conditions, in people with (n = 30) and without (n = 35) chronic LBP. Comparisons revealed more out-of-phase coordination amplitude (sagittal plane) in the LBP group across the full task (P = 0.04, eta η = 0.26) and both conditions (i.e., load vs. no load). Greater coordinative variability during the return motion of the task was also observed under both loaded (P = 0.005, eta η = 0.31) and non-loaded (P = 0.04, eta η = 0.28) conditions, in the LBP group (56). Similar behaviors have also been reported in the works of Lamoth et al. who showed greater coordinative variability and more out-of-phase coordination amplitude in the frontal plane during a walking task, albeit with different methodology (principal component analyses) (24). Pranata and colleagues used CRP analyses to compare trunk-hip coordinative variability in healthy, low LBP-related disability, and moderate-high LBP-related disability groups, during a lifting task. The authors reported more out-of-phase coordination amplitude in the high vs. low disability group (mean difference = 12.97°, P = 0.041); however, there were no differences in coordination amplitude or variability when comparing the high and low disability groups with healthy controls (77). Lastly, Williams et al. explored variability in people with acute and chronic LBP from the standpoint of irregularity of ROM-angular velocity plots, and broadly found more variability in select phases of ROM testing and a lifting task in people with chronic LBP, compared to people with acute LBP (78). Other authors have reported no differences in coordinative variability when comparing people with and without chronic LBP during a more complex task (rowing), using vector coding procedures (62).

Overall, there is some evidence of more out-of-phase and more variable behavior (i.e., "loose" control) in people with LBP; however, a lack of consistent findings and significant study heterogeneity make it difficult to draw firm conclusions.

2.5.3. Summary of coordinative amplitude and variability literature base and gaps in knowledge

Discrepancies in reports of coordination amplitude and coordinative variability in LBP are common in the literature (35). While there is evidence pointing to more in-phase and less variable coordination in people with LBP (particularly during gait in the transverse plane (71)), one cannot discount well-conducted studies which have reported contradictory results (56), sometimes within the same sample (24). Disentangling the literature in this area is challenging, in-part due to heterogeneity in task choice (e.g., bending vs. running), task partitioning (e.g., bending vs. re-extension periods), task conditions (e.g., changing velocity, adding load), and choice of methodology (e.g., CRP analyses vs. vector coding). In particular, studies examining sagittal plane tasks are lacking and context is required to better understand divergence in reported findings (e.g., more in-phase and less variable (16) vs. less in-phase and more variable (56)). Detailed analysis of novel sagittal plane task will expand the evidence base and bring some clarity to this area of research.

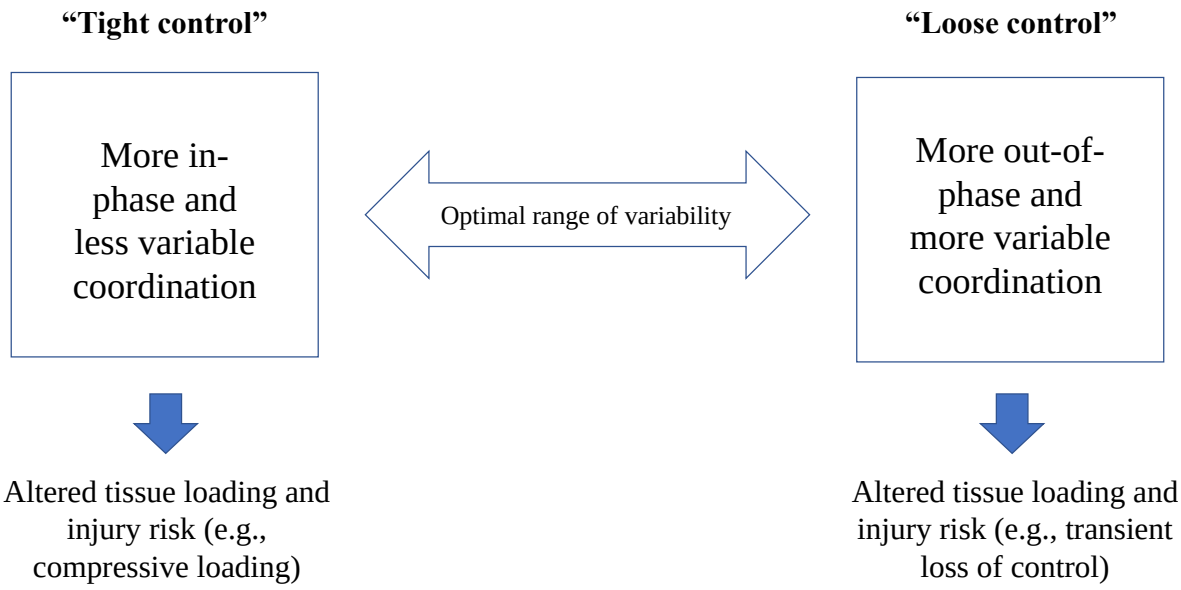

Figure 2.2. Coordination amplitude, variability, and musculoskeletal injury: The optimal range of coordinative variability and its implications for musculoskeletal injury based on proposed phenotypes of "tight" and "loose" control. Adapted from Hamill et al. 2012 and Van Diëen et al. 2018 (27, 43).

2.6. A biopsychosocial approach to motor behavior

As previously mentioned, the overall literature base studying coordination amplitude and variability in LBP is small compared to other aspects of motor behavior (i.e., spinal ROM and trunk muscle activity). Therefore, in order to discuss the relationship between biopsychosocial factors and coordination amplitude and variability properly, this work will first consider how biopsychosocial factors relate to the overarching construct of motor behavior. Following this, we will proceed to summarize the literature exploring relationships between biopsychosocial factors and coordination and variability in LBP.

2.6.1. Biopsychosocial factors and motor behavior

As outlined earlier in this chapter, biopsychosocial models are required to understand the complex relationship between pain and motor behavior in LBP. For instance, not only are changes in motor behavior diverse in presentation (6, 27), they have also been associated with pain and/or nociception (1), physiological changes to injured structures (79), plasticity of the nervous system (80), learned behaviors (81), and more. At a cognitive/psychological level, processes such as ongoing threat or fear of pain have been proposed to influence motor behavior, potentially serving as motivation to brace the trunk and protect the spine (i.e., greater fear leads to greater motivation for protection) (6, 82, 83).

Pain-related threat is broadly reflected in the constructs of pain-related fear (fear that emerges when stimuli related to pain are perceived as threatening) (82) and pain catastrophizing (an irrational, negative appraisal of pain) (84). These constructs are already important factors in clinical rehabilitation, having previously shown: (i) strong links with negative clinical outcomes in LBP (85, 86); (ii) having formed key aspects of important theoretical models for pain and

disability (e.g., fear-avoidance model (83)); and (iii) being modifiable targets in clinical rehabilitation of chronic LBP (87). Therefore, establishing the link between pain-related threat and motor behavior may serve to better inform our current understanding of, and improve treatment strategies for, chronic LBP (88). While our overall understanding of this topic is unclear, a few compelling studies help illustrate this relationship.

2.6.2. Evidence connecting pain-related threat and motor behavior

The relationship between pain-related threat and motor behavior can be studied by examining associations between the constructs of pain catastrophizing and/or pain-related fear, with biomechanical measures of motor behavior (trunk ROM, trunk muscle activity, spine coordination and variability). Past work reported a positive association between trunk muscle activity and catastrophizing scores in individuals with chronic LBP (n = 30) during a walking task (25). When controlling for pain levels and gait speed, this partial correlation was significant for 7/10 trunk muscles, with correlations coefficients ranging from 0.376 (right external oblique) to 0.532 (right rectus abdominis). This suggests that patients who catastrophize pain are more likely to guard their trunk during gait. Similarly, Geisser et al.'s work cross-sectionally linked greater pain-related fear with reduced lumbar flexion ($r = -0.55$, $P < 0.01$), greater erector spinae muscle activity ($r = -0.38$, $P < 0.01$) and smaller flexion-relaxation ratios ($r = -0.40$, $P < 0.01$) in people with chronic LBP (n = 76) (89). Using path-analysis, the authors found the relationships between greater pain-related fear and elevated trunk activity in both flexion ($\beta = 0.43$, $P < 0.01$) and extension ($\beta = 0.61$, $P < 0.01$) were mediated by reduced lumbar flexion (89). This suggests a direct link between greater pain-related fear and guarded motor behaviors, from the perspective of spinal ROM and trunk muscle activity. Other studies have shown a similar relationship in people

with LBP (90, 91), although, there are conflicting reports in the literature (92, 93). Thus, our overall understanding of this topic is unknown.

2.6.3. Evidence connecting pain-related threat and coordination amplitude and variability

The relationship between pain-related threat and coordination amplitude and variability is largely unknown. One recent study reported no relationship between fear and trunk kinematic variability in people with chronic LBP (n = 31) during gait (Hotelling's Trace F = 0.237; P = 0.396) (94). This study, however, examined variability of individual joint waveforms during a non-threatening task (walking) and used methods which may not align with coordination and variability from a Dynamic Systems Theory perspective. Otherwise, using noxiously induced back pain in healthy individuals, other authors have linked negative cognitions to reduced motor variability of postural strategies (in terms of deep abdominal muscle activity) (7) and increased local dynamic stability of the spine (via the Lyapunov exponent) (11, 95). How such findings translate to people with LBP, however, remains unclear.

In sum, there is preliminary evidence of a link between pain-related threat and motor behaviors consistent with guarding such as decreased spinal ROM and greater trunk muscle activity. This relationship, however, has yet to be fully explored from the perspective of coordination amplitude and variability.

2.7. Overall summary, gaps, and rationale

The burden of disease resulting from LBP is significant, and treatment outcomes remain unsatisfactory (12, 96). Changes in coordination amplitude and coordinative variability are thought to contribute to a cycle of pain and disability in LBP via abnormal loading of the spine. Despite

coordination and variability being increasingly recognized as an indicator of functional movement and healthy tissues (69), there are clear knowledge gaps regarding inconsistent observations of this feature in LBP (e.g., in-phase and reduced variability vs. out-of-phase and too much variability). The present review of the literature has highlighted: (i) methodological procedures, (ii) task conditions, and (iii) biopsychosocial factors, as elements which can influence observations of coordination amplitude and coordinative variability in people with LBP. This Ph.D. thesis will contribute evidence to current knowledge gaps in these areas.

First, greater pain-related threat may be linked to guarded motor behaviors, but the overall evidence base on this topic is unclear. Better insight into this relationship could have implications for clinical rehabilitation, where physical and psychological approaches to managing LBP are often siloed off as distinct interventions (97). A systematic synthesis of the literature base will determine if pain-related threat is related to guarded motor behaviors in people with LBP and determine the extent to which this relationship has been studied in the context of coordination amplitude and coordinative variability (chapter 3).

Next, discrepancies in findings of coordination amplitude and coordinative variability in LBP may reflect differences in task conditions and/or partitioning of tasks into separate movement periods. This is particularly true with respect to sagittal plane tasks where opposite findings have been reported in similar patient samples (16, 56). As such, we must expand our knowledge base by investigating coordination amplitude and variability during novel sagittal plane tasks, comparing to healthy individuals, and contribute to an evidence base that is relatively small (chapter 4).

Methodological procedures also underscore inconsistent reports of coordination amplitude and variability in LBP appreciably (55). While the Hilbert transform approach for CRP analyses

is a robust methodology, this procedure is prone to introducing end-effects (i.e., bias) in waveforms (66). This may be particularly problematic when one is concerned with key periods of movement (e.g., movement initiation). Thus, steps must be taken to improve methods used to measure coordination amplitude and coordinative variability in clinical populations, and empirical recommendations should be made to address this issue (chapter 5).

Lastly, to this author's knowledge, just one study has examined the relationship between pain-related threat and kinematic variability in LBP. As a result, this relationship has yet to be studied from the perspective of coordination amplitude and variability, and in a task more threatening than walking. With calls to conceptualize motor behavior from an integrated, biopsychosocial perspective (88), further research in this area is required and may provide context for inconsistent observations of coordination and variability in the literature (chapter 6).

2.8. Objectives and hypotheses

The overall purpose of this Ph.D. was to determine the impact of methodological, task-related, and biopsychosocial (i.e., pain-related threat) factors on coordination amplitude and coordinative variability in people with chronic LBP. This was achieved via four specific objectives/manuscripts.

Chapter 3: The relationship between pain-related threat and motor behavior in nonspecific low back pain: a systematic review and meta-analysis.

A starting point to inform this thesis was to investigate the overall relationship between guarded motor behaviors (including coordination amplitude and variability) and pain-related threat. The objective of this review was to determine the extent to which pain-related threat (via

pain-related fear and pain catastrophizing) was associated with motor behavior (via spinal ROM, trunk muscle activity, and trunk coordination and variability), in adults with non-specific LBP who were performing functional tasks. We hypothesized that measures of altered motor behavior consistent with guarding (e.g., reduced ROM, greater trunk muscle activity, less coordinative variability) would be associated with measures of elevated pain-related threat.

Chapter 4: **Movement variability in adults with low back pain during sit-to-stand-to-sit.**

Our research objective was to compare patterns of lumbo-pelvic coordination amplitude and variability, during repeated sit-to-stand-to-sit, in individuals with LBP and healthy adults. A secondary objective was to study the effect of task period (i.e., standing up vs. sitting down) on patterns of lumbo-pelvic coordination amplitude and variability. We hypothesized that participants with LBP would show more in-phase, less variable patterns of coordination for the lumbo-pelvic complex, in comparison with healthy controls, consistent with a protective behavior.

Chapter 5: The effects of data padding techniques on continuous relative-phase analysis using the Hilbert transform.

The objective of this chapter was to investigate the impact of three different data padding techniques (double reflection, mirroring, spline extrapolation) on end-effects following Hilbert-transformed CRP calculations, in comparison with extraneous real data, using lumbo-pelvic kinematic data when adults with LBP performed a sit-to-stand-to-sit task. We hypothesized that the double reflection padding method would result in the smallest deviations when compared to extraneous (i.e., real) data.

Chapter 6: The relationship between pain-related threat and coordination in adults with chronic low back pain during a lifting task

The objective of this chapter was to investigate the extent to which measures of pain-related threat (via pain catastrophizing and pain-related fear) were related to hip-lower lumbar and lower lumbar-upper lumbar inter-joint coordination amplitude and variability in adults with chronic LBP during a lifting task. A secondary objective was to determine if pain-related threat would differentiate between participants classified as "tight" and "loose" control, in terms of coordination amplitude and variability. We hypothesized that greater pain-related threat would be associated with "tight", or guarded coordination and variability.

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Preface to Chapter 3

People with LBP move differently than healthy individuals. These changes in motor behavior may cause increased pain during movement/exercise and negatively influence treatment outcomes for LBP. Chapter 2 outlined preliminary evidence linking biopsychosocial constructs suggestive of greater pain-related threat (e.g., pain catastrophizing) and guarded motor behavior. The overall state of the literature, however, is unknown. We hypothesize that greater pain-related threat may in-part explain observations of guarding in this group. Therefore, a starting point in this thesis was to summarize the relationship between pain-related threat and motor behavior, from the perspective of spinal mobility, trunk muscle activity, and coordination.

Chapter 3: The relationship between pain-related threat and motor behavior in non-specific low back pain: a systematic review and meta-analysis.

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3.1. Abstract

Objective: While pain-related fear and catastrophizing are predictors of disability in LBP, their relationship with guarded motor behavior is unclear. The aim of this meta-analysis was to determine the relationship between pain-related threat (via pain-related fear and catastrophizing) and motor behavior during functional tasks, in adults with LBP.

Methods: This review followed PRISMA guidelines. MEDLINE, Embase, PsychINFO and CINAHL databases were searched to April 2021. Included studies measured the association between pain-related fear or pain catastrophizing and motor behavior (spinal range of motion, trunk coordination and variability, muscle activity) during movement, in adults with non-specific LBP. Studies were excluded if participants were post-operative or diagnosed with specific LBP. Two independent reviewers extracted all data. The Newcastle-Ottawa Scale assessed for risk of bias. Correlation coefficients were pooled using the random-effects model.

Results: Reduced spinal range of motion during flexion tasks was weakly related to pain-related fear (15 studies, r = -0.21, 95% confidence interval = -0.31 to -0.11, P < 0.001) and pain catastrophizing (7 studies, r = -0.24, 95% confidence interval = -0.38 to -0.087, P = 0.002). Pain-related fear was unrelated to spinal extension (3 studies, r = -0.16, 95% confidence interval = -0.33 to 0.026, p = 0.093). Greater trunk extensor muscle activity during bending was moderately related to pain-related fear (2 studies, r = -0.40, 95% confidence interval = -0.55 to -0.23, P < 0.001). Pain catastrophizing, but not fear, was related to higher trunk activity during gait (2 studies, r = 0.25, 95% confidence interval = 0.063 to 0.42, P < 0.013). Methodological differences and missing data

limited robust syntheses of studies examining muscle activity, so these findings should be interpreted carefully.

Conclusion: We found a weak to moderate relationship between pain-related threat and guarded motor behavior during flexion-based tasks, but not consistently during other movements.

Impact statement: Future clinical research should build on these findings by exploring the advantages of integrated treatment strategies that target both psychological and motor behavior processes, compared to traditional approaches.

3.2. Introduction

Movement changes following pain and injury (1, 2). This change in motor behavior is understood to initially be protective, and reflect the nervous system's attempt to reduce actual or anticipated threat to the body/tissues (1, 2). In the spine, motor behavior is an overarching term used to describe the underlying biomechanics of movement, and can be quantified through measures of spinal range of motion (ROM), trunk coordination and movement variability, and muscle activity (Table 3.1). In chronic LBP, persistent guarded motor behavior such as restricted movement at the spine (3), excessive trunk muscle activity (e.g. bracing) (4, 5), and reduced variability in movement strategies (6), have all been observed during a variety of functional tasks (e.g., lifting, reaching). While this presentation is not uniform in all people with LBP, a subset of individuals adopt a guarded approach to movement (7, 8). Over time, these guarded behaviors are thought to sub-optimally load tissues, leading to pain, re-injury, and disability (1, 2).

Understanding how motor behavior is changed following injury is a complex task. For instance, not only are these changes diverse in presentation (1, 7), they have also been associated with pain and/or nociception (2), physiological changes to injured structures (9), plasticity of the nervous system (10), learned behaviors (11), and more. At a cognitive level, processes such as ongoing threat or fear of pain have been proposed as motivation to maintain guarded motor behaviors (1, 12, 13). Pain-related threat is broadly reflected in the constructs of pain-related fear (fear that emerges when stimuli related to pain are perceived as threatening) (12) and pain catastrophizing (an irrational, negative appraisal of pain) (14). These constructs have been studied in the context of the fear-avoidance model of pain (12, 13); and may help explain why guarding persists in some people with LBP.

The fear-avoidance model of pain proposes that greater pain-related fear and pain catastrophizing prompt hypervigilance and avoidance-type behavior, resulting in ongoing disability and other negative sequalae (12, 13). There appears to be inconsistencies in previous work linking pain-related threat and altered motor behavior (4, 15), and there is a need for a systematic synthesis of this literature base. Better understanding of this relationship may provide context for divergence in motor behaviors observed in LBP and help guide clinical interventions.

The aim of this review was to determine the extent to which pain-related threat (via pain-related fear and catastrophizing) is associated with motor behavior (via spinal ROM, trunk muscle activity, and trunk coordination and variability), in adults with non-specific LBP who are performing functional tasks. We hypothesize that measures of altered motor behavior consistent with guarding (e.g., reduced ROM, greater trunk muscle activity) will be positively associated with measures of pain-related threat.

3.3. Methods

The review protocol was registered with the international database of prospectively registered systematic reviews (PROSPERO, # CRD42020162337) and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (16).

Studies were included if they met the following criteria: (i) participants were adults (aged 18-65) with chronic or acute/sub-acute non-specific LBP (17); (ii) the study examined the relationship between a measure of motor behavior (e.g., spinal ROM) during a functional task (e.g., bending, walking) and pain-related fear or pain catastrophizing; (iii) motor behavior of the lumbar spine/trunk was quantified as spinal ROM, trunk muscle activity, trunk coordination and/or motor variability, and were measured using an objective tool (e.g., motion capture system); (iv)

pain-related fear was measured using the Tampa Scale for Kinesiophobia (TSK) (18) or TSK-11 (19), Fear-Avoidance Belief Questionnaire (FABQ) (20), Photograph series of daily activities (PHODA) (21) or the short electronic version (PHODA-Sev) (22), or Pain and Anxiety Symptoms Scale (PASS) (23); while pain catastrophizing was measured via the Pain Catastrophizing Scale (PCS) (24), or the Coping Strategies Questionnaire catastrophizing subscale (CSQ-Cat) (25).

Studies were excluded if: (i) participants were primarily diagnosed with specific LBP (e.g., radiculopathy, spondylolisthesis), had received surgical interventions to the spine, or had experimentally induced LBP (e.g., saline injection); (ii) the study participants' expectations were manipulated during the functional task; (iii) subjects were exposed to trunk perturbations or unsteady surfaces (e.g., reactionary reflexes, postural sway); and if (iv) the studies were a commentary/editorial, an unrelated review article, a case study, or conference abstract.

Acute/sub-acute LBP was defined as symptoms of less than twelve weeks in duration, while chronic LBP was defined as symptoms lasting greater than three months (26). Considering possible group differences in motor behavior based on stage of injury, this distinction was made to allow meaningful comparisons in our analyses. In the event a study combined individuals with acute/sub-acute and chronic LBP, the mean duration of symptoms was used to determine their stage of injury. Non-specific LBP was defined as pain primarily located between the gluteal folds and lower ribs, with or without leg pain, and via exclusion of a diagnosis of specific LBP (26). In the few cases where non-specific and specific LBP or surgical LBP were combined, a study was included if greater than 50% of the sample had non-specific LBP. In the case of longitudinal or interventional studies with multiple time points, baseline scores were extracted.

3.3.1. Data sources and searches

Four electronic databases (MEDLINE, Embase, PsycINFO, CINAHL) were searched on October 9th, 2019, using four main search strings: low back pain, functional tasks, pain-related threat, and trunk biomechanics (i.e., motor behavior). An information specialist from McGill University (Montreal, Canada) helped develop the search strategy. Searches were constrained to the English and French languages. Reference searches of all articles read in full were conducted. The search string used in MEDLINE is shown in Appendix 1.1. Titles and abstracts were independently screened by two reviewers (P.I., A.T.) and disagreements were resolved by a third party (S.R.). Full versions of relevant articles were obtained and independently reviewed (P.I., A.T.) for eligibility. In the event of uncertainty, a third reviewer (S.R.) was consulted, and a decision was rendered. A data extraction sheet was developed, piloted on five randomly selected articles, and refined accordingly. Two reviewers independently extracted data (P.I., A.T.). Disagreements were resolved through discussion and via a third reviewer (S.R.). In the event of missing information, study authors were contacted up to three times at two-week intervals to obtain missing data.

3.3.2. Data extraction and quality assessment

The following data were extracted: (i) authors and year of publication; (ii) study design and setting; (iii) number of participants and sample characteristics (age, sex, pain intensity, disability); (iv) definition of non-specific LBP; (v) duration and severity of symptoms; (vi) functional task; (vii) motor behavior outcome and measurement methodology; (viii) pain-related threat outcome and tool used; (ix) methodological approach for statistical analyses; (x) main findings linking motor behavior and pain-related threat outcomes with statistical result(s); (xi) funding source; and (xii) study conclusions.

An adapted version of the Newcastle-Ottawa quality assessment scale for cross-sectional studies was used to assess for risk of bias (27, 28). The Newcastle-Ottawa scale utilizes a scoring system which evaluates studies on (i) selection of study participants, (ii) comparability of study groups, and (iii) the exposure and outcome of interest. Because control groups were not relevant to our question, the comparability construct was adapted to reflect a study's decision to control for confounding factors (e.g., pain) during their analyses between threat and motor behavior. Given we are only interested in relationships between variables at baseline, this tool was also used to appraise any longitudinal studies. Studies were scored out of ten and categorized as very good (9-10 points), good (7-8 points), satisfactory (5-6 points) and unsatisfactory (0-4 points). Full details are available in the Appendix (Appendices 1.2 to 1.4). Two independent reviewers (P.I., A.T.) performed the appraisal and resolved inconsistencies through discussion. In the event of disagreement, a third reviewer was consulted (S.R.).

3.3.3. Data synthesis and analysis

Data were categorized by stage of LBP (acute/sub-acute vs. chronic), pain-related threat (pain-related fear, pain catastrophizing), and motor behavior (spinal ROM, trunk muscle activity, trunk coordination and variability). Outcomes for motor behavior were further sub-categorized by task and movement being performed (e.g., spinal flexion, gait). Separate meta-analyses were performed by pooling correlation coefficients across different studies with common outcomes for motor behavior using a random-effects model (29). Correlation coefficients reported as Pearson's *r* or Spearman's rho were analyzed together. If R^2 from simple linear regression or Kendall's Tau were reported, data were converted to Pearson's *r* and included in analyses (30). When correlations were unavailable, standardized regression coefficients were used in the analysis (31). As a

summary statistic, pooled correlations were presented with 95% confidence intervals (29). Strength of pooled correlations were interpreted as small ($r = 0.10$ to < 0.30), medium ($r = 0.30$ to < 0.50), and large ($r > 0.50$). Statistical significance was set at $P < 0.05$. Studies not suitable for meta-analysis (e.g., missing data, inappropriate statistical methodology) were described separately. All analyses were performed using Comprehensive Meta-Analysis (CMA Version 3, Englewood, NJ, USA).

If a study reported multiple related outcomes for motor behavior, an average effect size with adjusted variance was calculated as suggested by Borenstein (29). For example, Vaisy et al. reported separate correlations for pelvic and lumbar flexion ROM with pain-related fear (32). Given most studies considered the lumbo-pelvic region as a functional unit (e.g., L1-S2), an average effect size was calculated to improve comparability. Similarly, studies examining muscles performing the same action (e.g., erector spinae at L1 vs. L2) were combined into an average effect size. Studies reporting the same outcome for motor behavior (e.g., lumbar ROM) across similar tasks (e.g., bending and lifting) were combined. If a study reported multiple effect sizes (e.g., correlations of TSK and FABQ scores with lumbar ROM), a pre-determined decision-making process was used to determine the effect size used in our primary analyses. Given there is no preferred tool to measure pain-related fear (33), a hierarchy based on frequency of occurrence was used to determine the effect size used for analyses when multiple correlations were available (Appendix 1.5). Finally, the sign on the correlation coefficient was reversed for measures of finger-to-floor distance for spinal flexion ROM. This was done to ensure that negative correlations consistently reflected the relationship between greater threat (i.e., higher fear) and more guarded spinal ROM (i.e., reduced spinal ROM), across different studies.

Heterogeneity was described using the I^2 and T^2 statistics (34). Values of 25%, 50%, and 75%, correspond to low/mod/high relative heterogeneity, respectively. Heterogeneity was tested for significance using Cochran's Q ($P<0.10$).

Manual inspection of funnel plots was used to assess for publication bias across studies (29). When possible, acute/sub-acute and chronic LBP were analyzed separately (i.e., sub-group analyses). Given many different tools were used to measure pain-related fear and multiple correlations were often available in a single study, we employed a pre-determined selection process to determine which effect size was included in our analyses (Table 3.2, Appendix 1.5). To investigate if this may have influenced our findings, sensitivity analyses using upper and lower bound analysis was used (35). Specifically, if correlations between a single outcome for motor behavior and multiple pain-related threat outcomes measuring the same construct were available, the strongest correlations in the negative direction ("lower bound analysis") and in the positive direction ("higher bound analysis") were included in separate meta-analyses, regardless of tool used (35).

3.3.4. Role of the funding source

The funders played no role in the design, conduct, or reporting of this study.

Table 3.1. Measurement characteristics for motor behavior outcomes

Motor Behavior Outcome		Definition	Methodology/Tool Used	Number of outcomes
Spinal Range of Motion	*Lumbar flexion*	Sagittal plane lumbar joint angles	FFD, Goniometry, Motion capture, Potentiometer, not listed	22
	Lumbar extension	Sagittal plane lumbar joint angles	Motion capture, Goniometry	4
	Lumbar rotation	Transverse plane lumbar joint angles	Motion capture	3
	Lumbar side-flexion	Frontal plane lumbar joint angles	Motion capture	2
Trunk EMG	*FRR*	A measure of erector spinae's ability to relax at full spinal flexion, such that, greater ratios indicate less relative muscle activity	sEMG	5
	Muscle activity	An estimate of muscle activity and function using sEMG	sEMG	10
Coordination and Variability	*Coordi-nation*	Describes relative movement between joints or relative activity levels between muscles during functional tasks	Motion capture, sEMG	3
	Motor Varia-bility	Within-individual variability in movement patterns across multiple repetitions of a task, in terms of kinematic parameters or muscular activity.	Motion capture, sEMG	5

ROM: Range of Motion; FFD: Finger-floor distance; FRR: Flexion-relaxation ratios; sEMG: surface electromyography.

Table 3.2. Measurement characteristics for pain-related threat outcomes

Pain-Related Threat Outcome	Definition	Tool Used	Number of outcomes
Pain-related Fear	Fear that emerges when stimuli related to pain are perceived as threatening	TSK	16
		TSK-AA	1
		FABQ	5
		FABQ-PA	7
		PASS	1
Pain Catastrophizing	An irrational, negative appraisal of pain	PCS	8
		CSQ-Cat	3

TSK: Tampa Scale for Kinesiophobia; TSK-AA: Tampa Scale for Kinesiophobia Activity Avoidance subscale; FABQ: Fear-Avoidance Beliefs Questionnaire; FABQ-PA: Fear-Avoidance Beliefs Questionnaire Physical-Activity subscale; PASS: Pain Anxiety Symptoms Scale; PCS: Pain Catastrophizing Scale; CSQ-Cat: Coping Strategies Questionnaire

3.4. Results

A search conducted of Medline, Cinahl, PsycINFO and EMBASE retrieved 1524 records (October 2019). Forty-six additional records were identified through reference searching and by retrieving references from a similar systematic review (36). Once duplicates were removed, two independent reviewers screened 822 abstracts and selected 112 articles to be read in full. Eighty-two articles were excluded. An updated search conducted on April 21st, 2021, generated 85 additional abstracts. Of these, 18 articles were read in full and 4 were retained for our analyses. Authors of 8 articles were contacted for additional information. Thirty-three articles were retained for our review, and 21 were included in 5 separate meta-analyses (Figure 3.1). Publications not suitable for meta-analysis were reviewed qualitatively (n=12).

Key characteristics of included studies are listed in Table 3.3. Retained studies involved 1907 participants. Participants were coded as chronic LBP in twenty-seven studies (4, 5, 15, 32, 37-60), acute/sub-acute LBP in four (61-64), and distinct groupings of acute/sub-acute and chronic LBP in two (65, 66). Studies used a cross-sectional (n = 27), or longitudinal design (n = 6).

Of the included 33 studies, there were a total of 54 outcomes for motor behavior. This occurred because some studies reported multiple outcomes (e.g., pain-related fear with spinal ROM and trunk muscle activity) (15). These 54 outcomes were organized into the following categories: spinal ROM (n = 31), trunk muscle activity (n = 15), and coordination and movement variability (n = 8). Measurement characteristics of outcomes for motor behavior are listed in Table 3.1. Many studies used a functional task related to bending, lifting, or walking; while others investigated general spinal ROM. Based on our pre-determined selection process, 30 studies analyzed their motor behavior outcome(s) in relation to pain-related fear, and 11 in relation to pain catastrophizing (Table 3.2).

3.4.1. Risk of bias within studies

According to the Newcastle-Ottawa Scale, 18 of the 33 studies were classified as "good", 14 as "satisfactory", and 1 as "unsatisfactory" (Table 3.3). See Appendix for inter-rater agreement and full details (Appendices 1.2 to 1.4). No studies were considered as "very good." The most common risks of bias were related to failure to calculate sample size and to control for a primary confounder in their analyses (e.g., pain).

3.4.2. Motor behavior and pain-related threat: spinal range of motion

3.4.2.1. Pain-related fear and spinal flexion

Fifteen of eighteen studies examining a spinal flexion task (e.g., bending) were suitable for meta-analysis (Table 3.4, Figure 3.2A) (15, 32, 38, 40, 41, 43, 49, 51-53, 57, 61, 62, 64, 66). Pooled analyses found a small negative association between levels of pain-related fear and spinal flexion ROM in people with LBP ($r = -0.21$, 95% CI = -0.31 to -0.11, $P < 0.001$). This indicates that greater fear was related to reduced spine motion. Moderate heterogeneity was found ($I^2 = 59.0\%$, $P = 0.001$), suggesting some dispersion in our overall effect size unrelated to sampling error. Three studies reporting mixed findings were excluded from analysis due to missing data and/or differences in statistical methodology (Table 3.3) (46, 47, 63).

3.4.2.2. Pain-related fear and spinal extension

Three studies examining a spinal extension task were suitable for meta-analysis (32, 40, 41). Pooled analyses revealed no statistically significant association between pain-related fear and spinal extension in chronic LBP ($r = -0.16$, 95% confidence interval = -0.33 to 0.026, $P = 0.093$).

Heterogeneity was low ($I^2 < 0.001\%$, $P = 0.58$). (Table 3.4, Figure 3.2B). Missing data precluded synthesis of an additional study (64).

3.4.2.3. Pain-related fear and other tasks

Two studies were excluded from meta-analyses because they were the sole article to examine a task (40) or outcome (44). Jette et al. reported no statistically significant association between pain-related fear and both lateral flexion and rotation ROM of the lumbar spine (range $r = -0.35$ to 0.04, $P > 0.05$) during movement testing in people with chronic LBP (40). Similarly, Lamoth and colleagues found no statistically significant link between fear and trunk rotational amplitudes during gait ($P > 0.05$) (Table 3.3) (44).

3.4.2.4. Pain catastrophizing and spinal flexion

Seven studies examining spinal flexion were suitable for meta-analysis (32, 43, 49-51, 62, 65). Pooled analyses found a weak negative association between pain catastrophizing and spinal flexion ROM in people with chronic LBP ($r = -0.24$, 95% confidence interval = -0.38 to -0.087, $P = 0.002$) (Table 3.4, Figure 3.2C). This suggests that greater levels of catastrophizing are associated with reduced spinal flexion. Heterogeneity was low ($I^2 = 28.9\%$, $P = 0.20$).

3.4.2.5. Pain catastrophizing and spinal extension

One study reported no statistically significant link between pain catastrophizing and spinal extension ROM, in chronic LBP (range $r = -0.32$ to -0.20, $P > 0.05$) (32).

3.4.3. Motor behavior and pain-related threat: trunk muscle activity (EMG)

3.4.3.1. Pain-related fear and spinal flexion

Five studies examined flexion-relaxation ratios during full spinal flexion, although only two were suitable for meta-analysis (Table 3.4, Figure 3.3A) (15, 58). Pooled analyses of two studies found a moderate negative correlation between pain-related fear and flexion-relaxation ratios in people with chronic LBP ($r = -0.40$, 95% CI = -0.55 to -0.23, $P < 0.001$). This suggests a link between greater pain-related fear and smaller relaxation ratios (i.e., greater relative muscle activity during full flexion) in people with chronic LBP. Heterogeneity was low ($I^2 < 0.001\%$, $P = 0.361$). Two studies were not suitable for meta-analysis (missing data); however, both reported no statistically significant link ($P > 0.05$) between flexion-relaxation ratios and pain-related fear (46, 53). Efforts to obtain data were unsuccessful. The remaining study was not suitable for synthesis because they used the same sample as Geisser et al. and reported some identical outcomes (Table 3.3) (37). Their work examined a series of relaxation ratios and reported mixed findings (range $r = -0.48$ to $r = 0.02$).

3.4.3.2. Pain-related fear and muscle activity during gait

Three works reported little (42) or no statistically significant association between pain-related fear and trunk muscle activity during gait in people with chronic LBP (5, 44). Methodological differences (e.g., activity of erector spinae vs. rectus abdominis) and missing data, precluded synthesis of findings.

3.4.3.3. Pain-related fear and muscle activity during other tasks

Methodological differences and missing data did not allow synthesis of four studies (15, 39, 48, 55). During phases of flexion/re-extension of a bending task in chronic LBP, statistically

significant associations were reported between pain-related fear and internal oblique/transverse abdominis (range r = 0.61 to r = 0.82, P < 0.05) (48), erector spinae (range r = -0.40 to -0.38, P<0.01) (15), but not external oblique (P > 0.05) (48, 55). In contrast, no statistically significant correlations were reported between pain-related fear and erector spinae activity in a fully flexed position (15, 39), or with trunk extensors across five functional tasks (range r = -0.18 to r = 0.17, all P > 0.05) (4).

3.4.3.4. Pain catastrophizing and muscle activity during gait

Two studies examining pain catastrophizing and trunk muscle activity during gait were suitable for meta-analysis (54, 56). In people with chronic LBP, pooled analyses found pain catastrophizing was weakly associated with erector spinae activity (r = 0.25, 95% CI = 0.063 to 0.42, P = 0.013). This indicates that greater pain catastrophizing is related to greater erector spinae activity (Table 3.4, Figure 3.3B).

3.4.4. Motor behavior and pain-related threat: coordination and variability

Two studies reported no statistically significant relationships between pain-related fear and trunk coordination and variability during gait (44, 60), although synthesis was not possible. Others examining trunk coordination and variability during bending found inconsistent relationships with fear and catastrophizing (55, 59) (Table 3.3). Task and methodological differences precluded data synthesis.

3.4.5. Additional analyses

Publication bias was assessed using funnel plots for meta-analyses involving greater than 10 studies. In the sole case (pain-related fear and spinal flexion ROM), relative symmetry is

observed, suggesting that publication bias is not present (Appendix 1.6). Subgroup analyses (chronic vs. acute/sub-acute LBP) were only possible for the findings relating spinal flexion with pain-related fear. This revealed that the acute/sub-acute subgroup had a slightly larger effect (r = -0.25, 95% CI = -0.41 to -0.076, P = 0.005), than the chronic group (r = -0.20, 95% CI = -0.31 to -0.072, P = 0.002) (Appendix 1.7).

Sensitivity analyses including the strongest correlations (positive direction), regardless of tool used, produced a small, significant effect (r = -0.14, 95% confidence interval = -0.25 to -0.02, P = 0.021; I^2 = 69.8%, P < 0.001), albeit with high heterogeneity (Appendix 1.8A). In contrast, including the strongest correlations (negative direction), regardless of tool used for pain-related fear, produced a greater effect (r = -0.28, 95% confidence interval = -0.36 to -0.19, P < 0.001; I^2 = 51.4%, P = 0.009) (Appendix 1.8B). This suggests that the measure for pain-related fear influences interpretation of this relationship.

Figure 3.1. PRISMA flow diagram

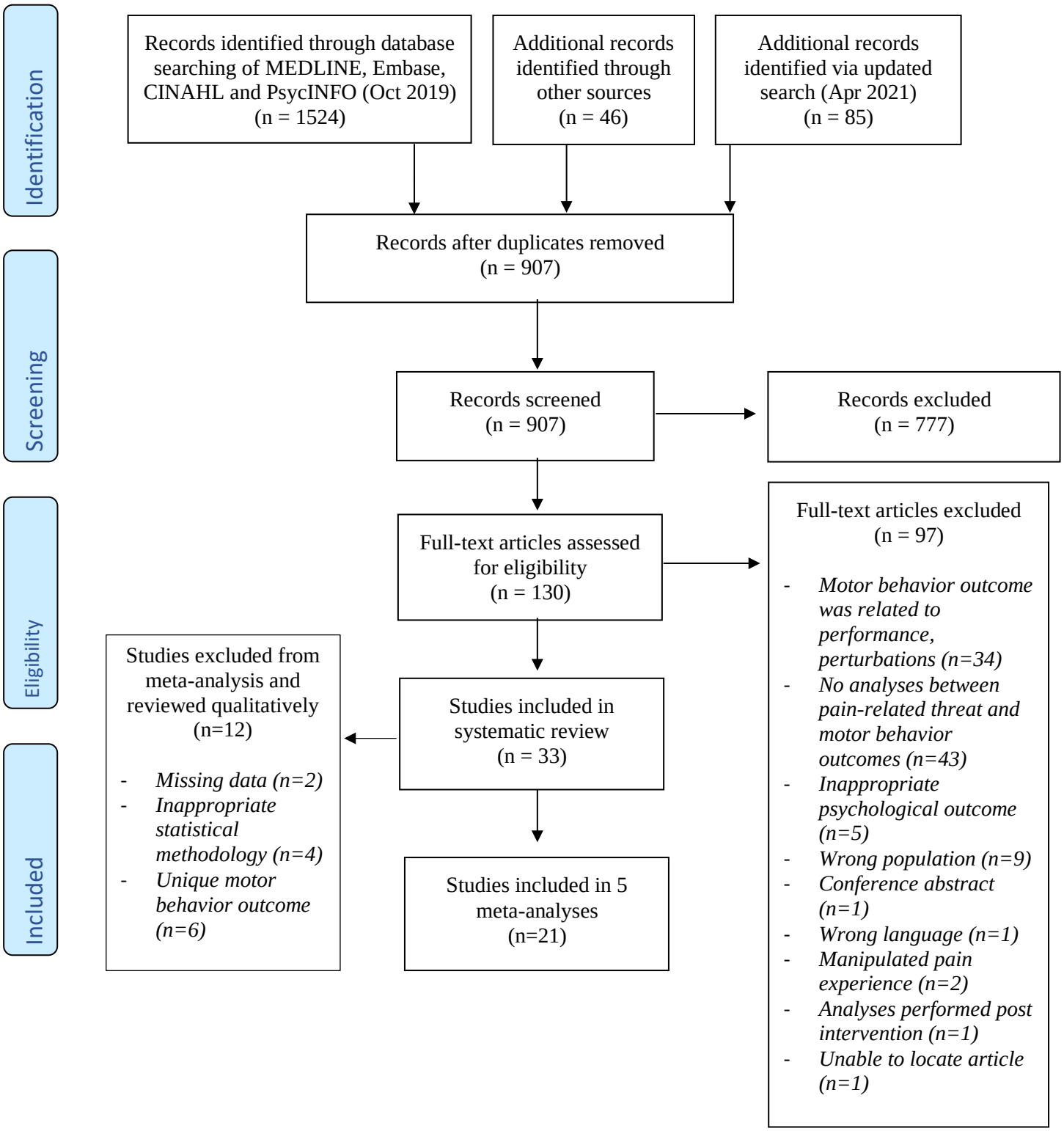


Table 3.3. Study characteristics

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
Alschuler et al. Cross-sectional	N: 76 M: 34 A: 40.6 (11.9)	C: SCLBP and NSCLBP (LBP > 3 months) D: 7.13 (8.65)	Spinal flexion	Trunk EMG (FRR at ES) M: sEMG on ES	TSK-13	6/10 S
*Alcaraz-Clariana et al. Case-Control	N: 33 LBP, 33 Con M: 22/20 A: 41.9 (14.8)	C: Acute/Sub-acute NSLBP (< 4 weeks) D: Not listed	Spinal flexion	Spinal ROM (lumbar flexion and extension; SF, Rot) M: Inertial measurement system (PSIS and Thoraco-lumbar junction)	TSK-11 FABQ FABQ-PA FABQ-W	8/10 G
Alsubaie et al. Cross-sectional	N: 15 LBP, 11 Con M: 9/8 A: 37.1 (9.1)	C: NSCLBP (>3 months, >3 days/week) D: Not listed	Spinal flexion Spinal extension	Motor variability (lumbar spine tracking variability) M: Motion capture system	FABQ-PA FABQ-W	7/10 G

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
*Demoulin et al. Cross-sectional	N: 50 M: 25 A: 44.2 (9.5)	C: CNSLBP (>3 months) D: 9.2 (9.8)	Spinal flexion	Spinal ROM (lumbar flexion) M: Finger to floor distance	TSK-tot Mean PHODA FVAS	7/10 G
Dubois et al. Cross-sectional	N: 52 M: 34 A: 39.7 (11.8)	C: CNSLBP (> half days over 12-month period) and Recurrent NSLBP (< half days over 12 month per year). D: 12.5 (10.3)	Spinal flexion	Trunk EMG (ES activity full flexion) M: sEMG on ES	FABQ, PCS, and STAI. Summari-zed using PCA	6/10 S
*Geisser et al. Cross-sectional	N: 76 M: 34 A: 40.6 (11.9)	C: SCLBP and CNSLBP (LBP > 3 months, dx by physiatrist) D: 7.13 (8.65)	Spinal flexion	Spinal ROM (lumbar flexion) Trunk EMG (FRR at ES) Trunk EMG (ES activity flexion and extension) M: sEMG on ES and goniometry	TSK-13	6/10 S

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
*George et al. Prospective case series	N: 63 M:26 A: 38.4 (10.2)	C: Acute/subacute NSLBP (<60 days) D: 27.7 days (16.4)	Spinal flexion	Spinal ROM (lumbar flexion) M: Inclinometer at T12/L1	FABQ-PA	5/10 S
*Grotle et al. Cross-sectional	N: 123 M: 55 A: 37.8 (10.4) N: 233 M: 106 A: 42.0 (8.9)	C: Acute/subacute NSLBP (<3 weeks) D: Not listed C: Chronic (> 1 year) D: Not listed	Spinal flexion	Spinal ROM (trunk flexion) M: Finger to floor distance	FABQ-PA FABQ-W	6/10 S
*Jette et al. Cross-sectional	N: 32 M: 13 A: 32.9 (7.83)	C: CNSLBP (> 3 months) D: Not listed	Spinal flexion Spinal extension	Spinal ROM (lumbar flexion and extension; RSF/LSF, RRot/LRot) M: Kinematics measured with Inertial measurement system (L1-S2)	FABQ-PA FABQ-W	7/10 G

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
*Kernan et al. Prospective series of consecutive cases	N:68 M:38 A:43 (10)	C: CNSLBP (back pain > 3 months, ODI >20) D: 2.33 (range: 0.25 to > 5)	Spinal flexion Spinal extension	Spinal ROM (lumbar flexion and extension) M: Single inclinometer at T12/L1	TSK FABQ-PA FABQ-W	6/10 S
*Kim et al. Cross-sectional	N: 15 Hi-LBP, 15 Lo-LBP, 15 Con M: 18/30 A: 23.0 (1.80)	C: Current or recurrent NSLBP > 7 weeks D: 1.87 (1.58)	Gait	Trunk EMG (ES, EO, RA, IO activity) M: sEMG on ES, EO, RA, IO	FABQ	8/10 G
*LaTouche et al. Cross-sectional	N: 30 Hi-SE, 30 Lo-SE M:25 A: 55.6 (13.0)	C: CNSLBP (>6 months) D: 6.10 (5.46)	Spinal flexion	Spinal ROM (lumbar flexion) M: Iphone goniometry (T12-S2)	FABQ TSK PCS	5/10 G
Lamoth et al. Cross-sectional	N: 22 CNSLBP, 17 Con M: 9/22 A: 38 (range 21-52)	C: CNSLBP (>3 months) D: 1.2 (range: 0.29 to 3.0)	Gait	Spinal ROM (trunk-pelvis rotations) Trunk EMG (ES activity) Coordination and motor variability (trunk-pelvis	TSK	6/10 S

Study Authors, design	Participant characteristics		Motor behavior characteristics		Pain-related threat tool	New-castle Ottawa Scale Score
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
				rotations and ES activity) M: Motion capture of thoracic, lumbar, pelvis, and sEMG ES		
*Lima et al. Cross-sectional	N: 40 M: 33 A: 35.2 (9.3)	C: CNSLBP (> 3 months) D: Not listed	Spinal flexion Sitting Standing Climbing a stair	Trunk EMG (MF, ESL, ESI activity) M: sEMG muscle activity	TSK	8/10 G
Mannion et al. Cross sectional	N: 148 M: 84 A: 44.8 (9.8)	C: CNSLBP >3 months, with or without referred pain (non-radicular) D: 10.2 (9.45)	Spinal flexion	Spinal ROM (lumbar flexion) Trunk EMG (FRR at ES) M: Potentiometer at sacrum and thoraco-lumbar junction and sEMG on ES	FABQ	5/10 S

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
Marich et al. Cross-sectional	N: 16 Lo-LBP, 16-Hi-LBP, 16 Con M: 16/32 A: 37.4 (18.6)	C: CNSLBP (> 12 months, > 1/2 days the year) D: 12.7 (7.2)	Spinal flexion	Spinal ROM (lumbar flexion) M: Visual 3D, Lumbar spine T12-S1.	FABQ-PA FABQ-W	8/10 G
Massé-Alarie et al. Cross-sectional	N:12 LBP, 13 con M: 6/12 A: 34.4 (13.1)	C: Lateralized CNSLBP (> 3 months) D: Not listed	Spinal flexion	Trunk EMG (IO/TrA, EO activity) M: sEMG at IO/TrA and EO	TSK	7/10 G
*Matheve et al. Cross-sectional	N: 55 M: 29 A: 41.1 (13.6)	C: CNSLBP (>3 months, >3 days/week) D: 5 (range 2-11)	Spinal flexion	Spinal ROM (lumbar flexion) M: Inertial measurement system L1-S1.	TSK-total TSK-AA TSK-SF PHODA-tot PHODA-lift PCS	8/10 G
*McCraken et al Cross-sectional	N: 30 M: 21 A: 40.9 (10.3)	C: CNSLBP (LBP >3 months) D: Median 2 (range (0.25-20.5)	Spinal flexion	Spinal ROM: (trunk flexion, mean ROM to point of pain tolerance) M: Not listed	CSQ-Cat	3/10 US

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
*Nieto-Garcia et al. Cross sectional	N: 30 M: 16 A: 49.3 (11.4)	C: CNSLBP (> 6 months) D: Not listed	Spinal flexion	Spinal ROM (lumbar flexion) M: Mobile goniometer device	TSK-11 TSK-AA TSK-SF PCS	5/10 S
*Osumi et al. Cross-sectional	N: 24 lo-fear, 21 hi-fear, 20 con M: not listed A: 56.3 (10.5)	C: SCLBP and NSCLBP (>6 months, pain >1/10) D: 11.5 (11.5)	Spinal flexion	Spinal ROM (lumbar flexion) M: Electro-goniometer at SP's of (T12-L2) and (S1-S3)	TSK-11 TSK-AA TSK-SF	8/10 G
*Pagé et al. Experimental cohort	N: 21 M: 13 A: 36.5 (11.8)	C: CNSLBP (>3 months) D: 7.36 (5.44)	Spinal flexion	Spinal ROM (lumbar and pelvic flexion) Trunk EMG (FRR at ES) M: sEMG on ES and motion capture for lumbar and pelvic angles	TSK	7/10 G
*Pakzad et al.	N: 15 lo-PC, 15 hi-PC, 15 Con M: 12/30	C: CNSLBP (>3 months, >2/10 pain, >12% ODI)	Gait	Trunk EMG (RA, EO, ESL, ESI, MF activity)	PCS	8/10 G

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
Cross-sectional	A: 33.3 (6.6)	D: 1.41 (0.81)		M: sEMG of RA, EO, ESL, ESI, MF		
*Salt et al. Cross-sectional	N: 13 Acute/ Subacute, 13 Chronic LBP M: 5 A: 53.5 (10.2)	C: Acute/Subacute NSLBP (<3 weeks), CNSLBP (>3 months) D: Not listed	Spinal flexion	Spinal ROM (pelvic flexion)	PCS	7/10 G
Svendsen et al. Cross sectional	N: 12 LBP, 12 Con M: 9/12 A: 38.6 (9.8)	C: CNSLBP (LBP <6 months) D: 0.38 (0.17)	Spinal flexion	Trunk EMG (ES, EO, activity) Coordination and motor variability (ES and EO activity) M: sEMG for EO and ES.	FABQ-PA FABQ-W CSQ-Cat	7/10 G
Thomas et al. Prospective series of consecutive cases	N: 18 Hi-fear and 18 lo-fear M: 13 A: 26.9 (6.9)	C: Acute/Subacute (LBP without radiculopathy at 3 weeks +/- 5 days)	Spinal flexion	Spinal ROM (lumbar flexion) M: Motion capture system (L1 relative to sacrum)	PASS	5/10 S

Study Authors, design	Participant characteristics		Motor behavior characteristics		Pain-related threat tool	New-castle Ottawa Scale Score
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
		D: 3 wks +/- 5 days)				
*Thomas et al. Prospective series of consecutive cases	N: 18 Hi-fear and 18 lo-fear M: 13 A: 26.9 (6.9)	C: Acute/Subacute (LBP without radiculopathy at 3 wks +/- 5 days) D: 3 wks +/- 5 days)	Spinal flexion	Spinal ROM (lumbar and thoracic flexion) M: Motion capture system (L1 relative to sacrum)	TSK PCS PASS	5/10 S
*Vaisy et al. Cross-sectional	N: 20 LBP, 19 Con M: 9/20 A: 32.9 (9.6)	C: CNSLBP (> 3 months, with symptom aggravation and remission in past 6 months, last >1 wk) D: 2.36 (2.36)	Spinal flexion Spinal extension	Spinal ROM (lumbar and pelvis flexion and extension; SF and Rot) M: Strain gauge measurement measured relative segment angles of lumbar and pelvis region	TSK-17 PCS	7/10 G
Van der Hulst et al. Cross-sectional	N: 63 LBP, 33 Con M: 33/63 A: 41 (11)	C: CNSLBP (>3 months of current or recurrent LBP)	Gait	Trunk EMG (ES activity) M: sEMG for ES	TSK-AA TSK-SF	6/10 S

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
		D: median 1.42 (range 0.25-6)				
*Van der Hulst et al. Cross-sectional	N: 63 LBP M: 33 A: 41 (11)	C: CNSLBP (>3 months of current or recurrent LBP) D: median 1.42 (range 0.25-6)	Gait	Trunk EMG (ES activity) M: sEMG for ES	CSQ-Cat	7/10 G
Veeger et al. Cross-sectional	N: 31 M: 10 A: 33 (IQR = 10)	C: CNSLBP (>3 months) D: median 3 (IQR = 4.9)	Gait	Motor variability (EO, IO, RA, ESL, ESI activity; trunk flexion, SF, Rot; pelvic posterior tilt, lateral tilt, and Rot) M: sEMG of EO, IO, RA, ESL, ESI and motion capture for pelvis and trunk	TSK	8/10 G

Study Authors, design	**Participant characteristics**		**Motor behavior characteristics**		**Pain-related threat tool**	**New-castle Ottawa Scale Score**
	- Participants (N) *- Sex (M)* *- Mean Age (A), (Standard deviation)*	*- Condition (C)* *- Duration (D,yrs)*	*Task classification*	*Outcome of interest and Methodology (M)*		
*Vincent et al. Cross-sectional	N: 100 non-obese, 92 obese M:102 A: 48.3 (18.4)	C: CNSLBP and SLBP (LBP for >3 months) D: 3.5 (7.25)	Spinal flexion	Spinal ROM (lumbar flexion) M: ROM using inclinometer	TSK-11	7/10 G
*Watson et al. Interventional cohort	N: 36 M: 15 A: 43.7 (9.3)	C: CNSLBP (>6 months) D: 4.6 (4.2)	Spinal flexion	Trunk EMG (FRR at ES) M: sEMG on ES	FABQ	6/10 S

*denotes study included in meta-analysis (n=21)

SCLBP: Specific Chronic Low Back Pain; CNSLBP: Chronic Non-Specific Low Back Pain; NSLBP: Non-Specific Low Back Pain; CLBP: Chronic Low Back Pain; LBP: Low Back Pain; Con: Control; FRR: Flexion Relaxation Ratio; FRP: Flexion Relaxation Phenomenon; Flex: Flexion; Ext: Extension; RSF: Right side flexion; LSF: Left side flexion; RRot: Right rotation; LRot: Left rotation; ES: Erector spinae; EO: External oblique; RA: Rectus abdominus; IO: Internal oblique; TrA: Transverse abdominus; MF: Multifidus; ESL: Erector spinae longissimus; ESI: Erector spinae iliocostalis; sEMG: surface Electromyography; ROM: Range of Motion; SP: Spinous process; PCA: Principle Component Analysis; ODI: Oswestry Disability Index; SE: Self-efficacy; TSK: Tampa Scale for Kinesiophobia; TSK-AA: Tampa Scale for Kinesiophobia

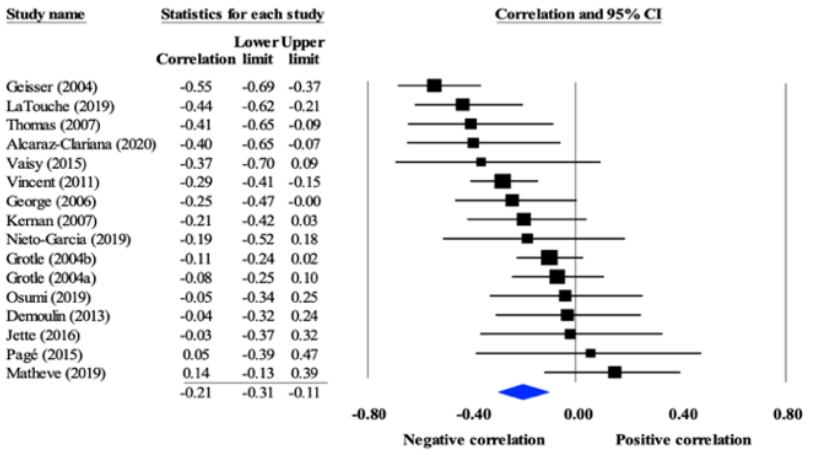
Activity Avoidance subscale; TSK-SF: Tampa Scale for Kinesiophobia Somatic Focus subscale FABQ: Fear-Avoidance Beliefs Questionnaire; FABQ-PA: Fear-Avoidance Beliefs Questionnaire Physical-Activity subscale; FABQ-W: Fear-Avoidance Beliefs Questionnaire Work subscale; PASS: Pain Anxiety Symptoms Scale; PHODA-Sev: Photograph Series of Daily Activities Short Electronic version; PHODA-lift: Photograph Series of Daily Activities Lifting image; FVAS: Fear Visual Analog Scale; PCS: Pain Catastrophizing Scale; CSQ-Cat: Coping Strategies Questionnaire Catastrophizing subscale; G: Good; S: Satisfactory; US: Unsatisfactory

Table 3.4. Summary of meta-analyses examining the relationship between pain-related threat and motor behavior in people with low back pain

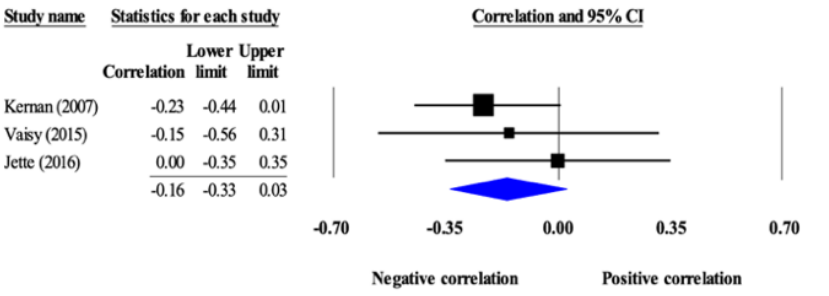
Pain-related threat	Motor behavior	No. studies	N	I^2 (%)	Correlation (95% CI)	P
Pain-related fear	*Spinal flexion ROM*	15	1137	59.0	-0.21 (-0.31 to -0.11)	**< 0.001**
	Spinal extension ROM	3	120	< 0.01	-0.16 (-0.33 to 0.026)	0.093
	FRR	2	112	< 0.01	-0.40 (-0.55 to -0.23)	**< 0.001**
Pain Catastrophizing	*Spinal flexion ROM*	7	257	28.9	-0.24 (-0.38 to -0.087)	**0.002**
	ES activity during gait	2	93	< 0.01	0.25 (0.063 to 0.42)	**0.013**

N: Number of participants; CI: Confidence Interval; ROM: Range of Motion; FRR: Flexion-Relaxation Ratios; ES: Erector Spinae. Bolded P-values indicate statistical significance.

A - Pain-Related Fear and Spinal Flexion



B - Pain-Related Fear and Spinal Extension



C - Pain Catastrophizing and Spinal Flexion

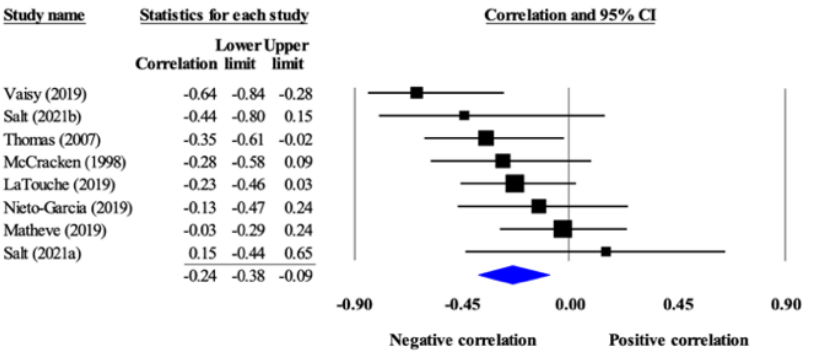
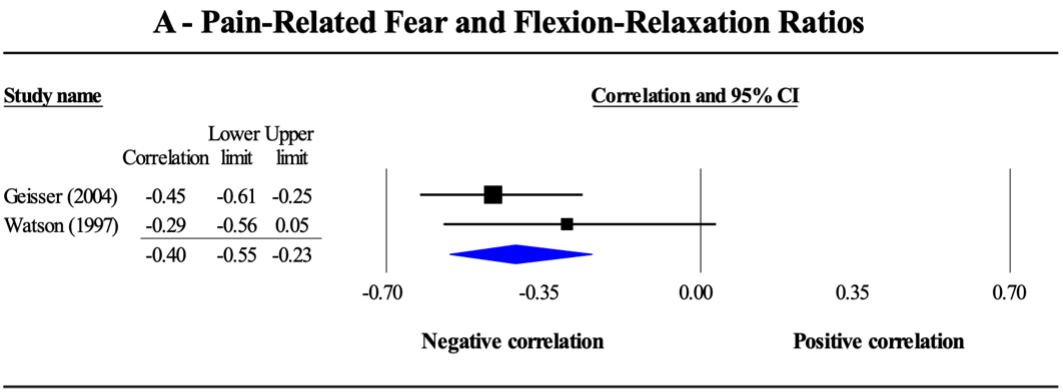


Figure 3.2. Forest plots for pain-related threat and spinal range of motion: (A) - Forest plot of 15 studies examining the relationship between spinal flexion range of motion and pain-related fear in people with acute/sub-acute and chronic low back pain, during flexion tasks; (B) - Forest plot of 3 studies examining the relationship between spinal extension range of motion and pain-related fear in people with chronic low back pain, during extension tasks; (C) - Forest plot of 7 studies examining the relationship between spinal flexion range of motion and pain catastrophizing in people with chronic low back pain, during flexion tasks.



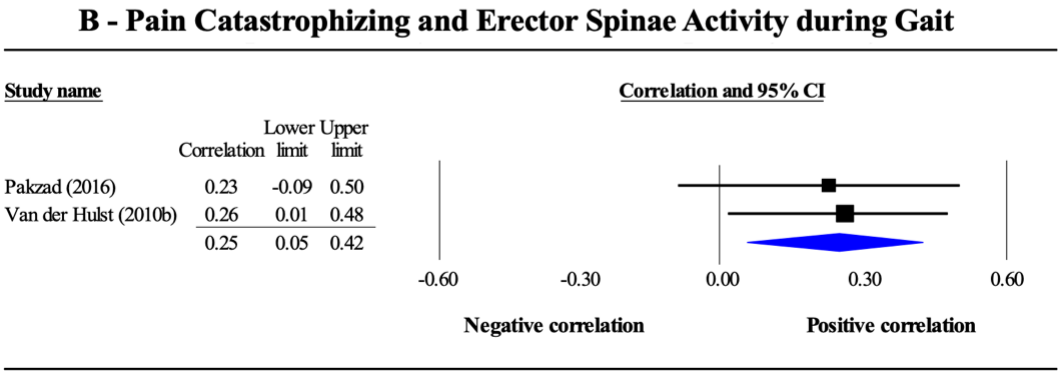


Figure 3.3. Forest plots for pain-related threat and trunk muscle activity: (A) - Forest plot of 2 studies examining the relationship between flexion relaxation ratios of erector spinae in full flexion, in people with chronic low back pain; (B) - Forest plot of 2 studies examining the relationship between erector spinae muscle activity and pain catastrophizing while walking, in people with chronic low back pain.

3.5. Discussion

This meta-analysis summarized the evidence linking pain-related threat and motor behavior during functional tasks in people with LBP. Findings were based on 33 studies of good (n=18), satisfactory (n=14), and unsatisfactory (n=1) quality, and suggest that (i) greater pain-related fear and catastrophizing are weakly associated with reduced lumbar flexion ROM, (ii) greater pain-related fear is moderately correlated with elevated trunk extensor muscle activity during bending (via relaxation ratios), (iii) greater pain catastrophizing is weakly associated with higher trunk extensor activity during gait, and (iv) fear was not significantly related to spinal extension ROM, nor motor variability during gait. Overall, the observed effects in the present study were of a smaller magnitude than previously reported effects that have examined the relationships between pain-related threat and other pain-related outcomes (e.g., disability, range r = 0.33 to 0.45) (67). Together, these findings have important implications for the clinical management of LBP and future research in this area.

Pain-related threat was associated with guarded motor behavior during flexion-based tasks, but not during extension or gait. There may be a few explanations. First, there are pervasive negative beliefs regarding bending in both LBP patients and health-care professionals (68, 69). Considering people with LBP often view their spine as ‘vulnerable’ and in need of protection (68, 70), it is logical that individuals experiencing pain-related threat may selectively stiffen their spines during tasks perceived as harmful (13). Supposedly less threatening tasks (e.g. extension), however, may not have been sufficient to prompt a guarded motor response. The lone exception was during gait. Greater trunk activity during gait is common in LBP (5, 44, 54), and while our analyses found elevated trunk extensor activity was linked to pain catastrophizing, this was not the case for pain-related fear (5, 44). Considering both the strong links between pain-related fear and

catastrophizing, and fear's negative association with performance measures of gait in LBP (71), the lack of other significant relationships in this review was somewhat surprising. This may reflect the complexity of trunk musculature, in which motor adaptations to pain are highly heterogeneous (2). Alternatively, this may point to catastrophizing and pain-related fear as having distinct relationships with motor behavior.

Twelve studies reported multiple effects for pain-related fear and motor behavior (e.g., TSK and FABQ correlations with spinal flexion). Considering there is no preferred tool for measuring fear (33), we employed a pre-determined decision-making algorithm to determine which effect was included in our analyses (Appendix 1.5). An interesting observation, however, were the differences in effect sizes between global (e.g., TSK) and 'task specific' (e.g., Phoda-lift) measures of fear when analyzing the same task. For example, Matheve et al. tested the impact of various measures for pain-related fear on flexion ROM and reported β = 0.14 and β = -0.35 for TSK, and Phoda-Lift, respectively (49). In this case, a task-specific measure for fear showed a considerably higher effect than a global measure. This highlights that tool selection impacts the interpretation of the relationship between fear and motor behavior appreciably, a finding corroborated by our sensitivity analyses (Appendix 1.8). Task-specific measures likely better reflect pain-related fear in relation to a specific task and may be a more ecologically valid tool to use when examining these relationships.

It remains unclear why guarded behaviors persist in some individuals with LBP. Past work, however, has hinted at pain-related fear and/or catastrophizing's capacity to predict motor behaviors consistent with guarding (72-74). While these works do not show causation, when considered alongside our results, the collective findings provide room to reflect on how threat and motor behavior are intertwined. First, threat could influence movement, indirectly, via pro-

nociceptive modulation of the nervous system (75, 76). When considering pain and nociception's direct link to motor behavior (1, 2), increased sensitivity to sensory inputs might facilitate nociception, pain, and guarding in LBP. Alternatively, an individual experiencing greater pain-related threat may learn to stiffen their spine as a perceived safety mechanism, regardless of the actual safety of this behavior or its impact on nociception (11). In turn, this learned behavior may result in invariable motor strategies, reduced proprioceptive input, and downstream cortical reorganization, re-enforcing a guarded behavior (10, 11, 77). Threat may also impact motor behavior by influencing activity in brain regions responsible for movement planning and execution (i.e., primary motor cortex, M1). For instance, exploratory work has connected pain-related fear and decreased corticomotor excitability (78); findings which build on preliminary evidence linking the motor system and cortical regions associated with threat (79). Lastly, one should consider the possibility of a bi-directional relationship between motor behavior and threat (80). For example, tissue injury may preclude an individual from generating a mechanically efficient movement strategy, and result in abnormal tissue loading and pain. Over time, repeated exposure to a painful activity may be sufficient to increase the threat value of pain associated with movement (e.g., painful bending leads to fear of bending). Overall, the relationship between threat and guarding requires further study. Prospective research examining the mechanism and direction of change in a clinical population may help flesh out this relationship.

While our data suggest a relationship between motor behavior and pain-related threat, this is it at odds with the sometimes siloed approach that has been previously used to manage movement-related and psychological factors in people with LBP (81). For example, graded exposure is an effective treatment approach grounded in the fear-avoidance model that targets elevated pain-related threat via simple exposure to feared activities (e.g. bending) (82). However,

this approach is not designed to consider the quality of motor behavior during exposure, and is designed to disregard potential nociceptive inputs during exposure tasks (e.g., nociception associated with tissue loading) (2). Not only are guarded motor behaviors often present in LBP (3, 8), recent work has shown that changes in such behaviors (e.g., restoring functional spinal ROM) were often strongly related to changes in pain and activity outcomes in some, but not all, individuals (83). Taken together, overlooking the contributions of guarded motor behavior may be a missed opportunity to improve the effectiveness of graded exposure interventions. On the other hand, treatment approaches targeting 'problematic' motor behaviors in isolation are discounting the significant negative influence of pain-related threat on patient and treatment outcomes (84). By overlooking fearful patient beliefs in the context of movement (e.g., that painful activity is perceived to contribute to further damage (85)), clinicians may discount key barriers preventing a person in pain from restoring functional movement and engaging in physical activity. Through this lens, extending traditional physiotherapy interventions targeting movement impairments to include educational strategies addressing unhelpful patient beliefs would be warranted. The emergence of integrated treatment approaches such as Cognitive Functional Therapy (CFT) – which target both the restoration of functional movement/postures and underlying cognitive and emotional factors – may offer an opportunity to better manage LBP (86). Recent works have shown CFT is effective at reducing disability (87), psychological outcomes (88), and preliminary evidence suggests it also restores changes in spinal mobility and flexion-relaxation (83). While promising, further work is required to determine the relative effectiveness of an integrated approach, such as CFT, compared to more traditional psychological or movement-based interventions.

Christe et al. published a similar review during the completion of the current manuscript, investigating the relationship between psychological factors (e.g., fear, depression) and motor

behavior in LBP (36). Our findings are largely in-line with theirs, pointing towards a small, but statistically significant relationship between pain-related fear, catastrophizing, and guarded motor behavior. Importantly, consistent findings through independent research helps add credibility to the collective findings. Our combined effects, however, were slightly larger – particularly for motor behavior during bending – albeit with greater heterogeneity. This may relate to the inclusion of new publications in our analyses which were not available at the time of their review (n = 8); subtle differences in study inclusion criteria regarding sample (e.g., we excluded studies including a majority of post-operative LBP cases and/or elderly subjects), and differing success in obtaining missing data.

These findings should be interpreted while considering certain limitations. First, the cross-sectional nature of our studies precludes determination of causation. Second, analyses on EMG data were performed on few studies and should be interpreted carefully. Methodological differences prevented robust syntheses of EMG data and made qualitative interpretation of these findings challenging. Fourth, sample characteristic varied considerably between studies. The categorization of individuals with LBP by stage of injury was somewhat arbitrary, especially when considering some samples were chronic and highly disabled, while others were chronic but highly functional. Also, degenerative spinal changes are common in LBP and may be linked to a stiffer spine (89). While our inclusion criteria (< 65 years old) likely helped limit this impact, we can't discount the potential influence of structural changes on the relationship between threat and motor behavior. Finally, we did not test pain as a confounder in the relationship between threat and motor behavior; however, recent work has shown that pain intensity and psychological factors impact motor behavior independently (36).

In summary, LBP is a heterogenous condition and many factors are capable of influencing motor behavior in this population. We found a weak to moderate relationship between elevated pain-related threat and guarded motor behavior during flexion-based tasks, but not consistently during other movements. Future clinical research should build on these findings by exploring the advantages of integrated interventional strategies that target both psychological and motor behavior processes, compared to more traditional siloed approaches.

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Preface to Chapter 4

Chapter 3 confirmed that there was sparse literature to report on the relationship between pain-related threat and joint coordination and variability. Given the emergence of joint coordination and variability as a potential factor influencing pain and injury in people with LBP, this was seen as a significant knowledge gap. That being said, chapter 2 outlined the inconsistencies in findings of coordination and variability in LBP (i.e., more in-phase and less variable vs. more out-of-phase and more variable). Therefore, prior to exploring the relationship between pain-related and coordination in this group, it was first necessary to establish a baseline and compare coordination in people with and without LBP, during a novel task.

Chapter 4: Movement variability in adults with low back pain during sit-to-stand-to-sit

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4.1. Abstract

Background: Differences in movement variability may be related to a guarded response to pain or a less robust movement pattern, indicating a potential dysfunction in motor control. The study objective was to compare patterns of lumbo-pelvic coordinative variability, during repeated sit-to-stand-to-sit, in individuals with LBP and healthy adults.

Methods: Participants were adults with LBP (n = 16) and healthy controls (n = 21). Kinematics for the T12-L3, L3-S1, and hip segments were measured using electromagnetic motion capture during 10 sit-to-stand-to-sit trials. Continuous relative phase analysis using the Hilbert transform method determined coordination and variability of the Hip-L3S1, and L3S1-T12L3 segments, deconstructed into 4 periods (start/up/down/end). T-tests compared coordination and variability of the full task between groups, and a mixed ANOVA compared the effects of group and period for the two segments.

Findings: Across the full task, the LBP group demonstrated more variable (mean difference = -6.95, 95% CI = -12.3 to -1.59) and greater out-of-phase behavior (mean difference = -22.6, 95% CI = -39.1 to -6.03) in the LHip-L3S1 segment. Group-period interaction effects revealed greater variability in the start period (mean difference = -0.325, 95% CI=-0.493 to -0.156) and more out-of-phase behavior in the start (mean difference = -0.350, 95% CI = -0.549 to -0.150) and end (mean difference = -0.354, 95% CI = -0.602 to -0 .105) periods for the LHip-L3S1 segment.

Interpretation: Excessive variability may relate to reports of poor spinal proprioception in LBP; however, based on our sample characteristics (low pain and disability) and lack of symptoms during the task, classifying our findings as dysfunctional may not be fully warranted.

4.2. Introduction

People with LBP move differently than pain-free individuals (1). Changes in movement variability, in particular, may indicate altered adaptability of the motor control system (2, 3). Decreased variability, as reported in chronic LBP during running (4) and bending (5), is consistent with a protective behavior in the presence of pain or when pain is anticipated (6) and may lead to repetitive tissue stress over time (7). Increased variability, as has been observed during reaching (8) and gait (9) in individuals with chronic LBP, may indicate error or noise in proprioceptive feedback and/or imprecise motor commands, reducing the robustness of the system (10). Movement variability in common functional tasks, therefore, may provide insight into the presence of motor control deficits in individuals with LBP.

Rising from a chair is a basic functional task, performed on average 60x/day (11), for which the lumbar spine accounts for 56-64% of mobility (12). Proper execution of this task necessitates coordinated behavior of the lumbo-pelvic complex. In healthy individuals, movement is initiated by simultaneous flexion of the spine/hips (1:3 ratio), followed by concurrent extension of the spine/hips to complete the process of standing upright (13). Shum et al. (2005) used more robust methodology (i.e., phase analysis) to examine inter-joint coordination during sit-to-stand-to-sit (STS) and found the lumbar spine leads the hips during the early phase of sit-to-stand and to lag behind the hips during upright standing; while the reverse pattern was observed during the stand-to-sit aspect of the task.

Amongst individuals with LBP, difficulty with this task is listed as a common functional complaint (14). Additionally, there are reports of reduced sagittal plane angles and altered lumbo-pelvic coordination during STS in individuals with chronic (15) and sub-acute LBP (16),

respectively. To our knowledge, however, movement variability during this task has not been investigated in this population.

Our research objective was to compare patterns of lumbo-pelvic coordination and inter-joint coordinative variability, during repeated sit-to-stand-to-sit, in individuals with LBP and healthy adults. A secondary objective was to study the effect of task period (i.e., standing up vs. sitting down) on patterns of lumbo-pelvic coordination and inter-joint variability. We hypothesized that participants with LBP would show more in-phase, less variable patterns of coordination for the lumbo-pelvic complex, in comparison with healthy controls, consistent with a protective behavior.

4.3. Methods

4.3.1. Participants

The study design was cross-sectional, with participants divided into a LBP group (n = 16) and a healthy group (n = 21). Participants were recruited from medical and rehabilitation clinics in Montreal, Quebec, and from the community. The sub-acute and chronic LBP group ($\bar{x}$ duration = 109.9 months, standard deviation (SD) = 113.5) had pain primarily located between the gluteal folds and ribs, with no evidence of spinal stenosis or radiculopathy, any serious underlying condition (cauda equina syndrome, cancer, infection), or another specific spinal condition (vertebral compression fracture, ankylosing spondylitis). Additional exclusion criteria for both groups included: neurological or respiratory conditions that might affect STS, major postural abnormality (e.g., scoliosis of $\geq 7^{o}$), previous spinal surgery or trauma (e.g., spinal fracture), or pregnancy in the past 2 years. Subject demographics were recorded on intake, and baseline scores for pain intensity, disability, and prognostic risk-factors for LBP-related disability were measured

using an 11-point numerical pain rating scale (17), the Oswestry disability index (18), and the STarT Back Screening Tool (19) respectively in participants with LBP (Table 4.1). Informed consent was obtained from participants and ethical approval for this study was received from the local research ethics board.

4.3.2. Sit-to-stand-to-sit (STS) task

Participants sat on a height-adjusted seat, with no back or arm support, such that their thighs were horizontal, their lower legs were vertical, and both feet were flat on the floor. Participants were then asked to adopt their preferred sitting posture, and to keep their arms crossed over their chest during the task. No further movement constraints were imposed. The STS task involved having the participants stand upright, from sitting, and return to sitting, as quickly as possible. A total of 10 trials were completed by each subject.

4.3.3. Data acquisition and analysis

Kinematic data were acquired in three dimensions, using an electromagnetic TrakSTAR motion capture system with model 800 sensors (Ascension Technology, Milton, VT, USA), sampled at 200 Hz. Manufacturer-reported accuracy for position and orientation, for each sensor, is 1.4 mm root mean square (RMS) and 0.50^{o} RMS, respectively. The sensors were mounted on the participants' skin, over the lateral side of the thighs (bilaterally), the base of the sacrum (S1), and the spinous process of the third lumbar (L3), and twelfth thoracic (T12) vertebra, using custom-molded urethane clips and double-sided tape.

A force plate (BP400600 NC, AMTI, Watertown, MA, USA) was mounted on the seat to capture surface reaction forces under the thighs and buttocks, sampled at 200Hz. This data was

used to determine the position of the center of pressure (CoP) at the support surface in order to identify STS events.

Kinematic and force plate data were smoothed using a low-pass, fourth-order, bidirectional Butterworth filter, with a cut-off frequency of 10 Hz. Orientation data for each sensor was used to create a [3 × 3] rotation matrix for each segment: thighs, S1, L3, and T12. Multiplying the rotation matrix for one segment by the inverse of the rotation matrix of an adjacent segment allows for the extraction of inter-segmental joint angles (20). Sagittal plane joint angles were calculated for: (i) the left hip - LHip (left thigh relative to S1); (ii) the lower lumbar spine - L3S1 (L3 relative to S1); (iii) and the upper lumbar spine - T12L3 (T12 relative to L3).

Each STS trial was divided into 4 sequential periods (start, up, down, end). The "start" period began at the point of greatest backward motion of the CoP at the seat surface (21), and ended at the point of loss of contact with the seat, as indicated by a vertical reaction force of < 10 N. The "up" period extended from the end of "start", to the position of upright standing as defined by maximal left hip extension. The "down" period was from upright standing to the point of contact of the participant with the seat, as measured by the initial recording of a vertical reaction force (>10 N). Finally, the "end" period extended from the point of contact with the seat to the point of greatest backwards motion of the CoP at the seat surface. Each STS trial was time normalized to 101 data points. Each period was also separately time normalized to 101 data points.

4.3.4. Relative phase and coordination analysis

The phase angle of each joint was determined using the time-normalized signals, x(t), and their Hilbert transform, $H(t) = H(x(t))$, as described by Lamb & Stockl (2014) (22). First, the amplitude of the data was centered around zero (Eq. 1).

Eq 1. $X_{centered}(t_i) = x(t_i) - min(x(t)) - (max(x(t)) - min(x(t)))/2$

Each signal was then transformed into a complex analytical signal, $\zeta(t)$, using the Hilbert transform, where the H(t) of x(t) serves as the imaginary portion of the analytical signal (Eq. 2).

Eq 2. $\zeta(t) = x(t) + iH(t)$

The Hilbert transform approach allows for a clear assessment of the phase difference between two arbitrary, non-stationary, non-sinusoidal signals. At any point in time, the phase angle of a joint could be calculated as the inverse tangent of the transformed signal $H(t_i)$ divided by the measured signal x(t) (Eq. 3). In order to address issues arising with data distortion (i.e., end effects) associated with a Hilbert transform, a data reflection method was used to pad the signal for each individual period.

Eq 3. $\phi(t_i) = arctan((H(t_i)/x(t_i)))$

Continuous relative phase (CRP) analysis was then used to describe patterns of inter-joint coordination (i.e., lag between two joints during movement). This was performed by determining the absolute difference in the phase angles for two adjacent joints (e.g., Hip and L3S1) across an entire movement cycle. Specifically, coordination was quantified by subtracting the phase angle of the distal joint from that of the proximal joint, generating CRP values for (i) LHip-L3S1 and (ii) L3S1-T12L3 segments, across each trial and period (Eq. 4). A value of 0 indicates that the two

joints are moving fully in phase with one another; while a value of 180 represents an anti-phase, or fully out-of-phase coupling (23).

Eq 4. $CRP(t_i) = \left| \phi_1(t_i) - \phi_2(t_i) \right|$

4.3.4.1. Inter-joint coordination amplitude: Mean Absolute Relative Phase (MARP)

To extract a value describing inter-joint coordination (i.e. in-phase/out-of-phase coupling relationships) for both the LHip-L3S1 and L3S1-T12L3 segments, the mean absolute relative phase (MARP) was calculated (24). First, ensemble curves were derived by averaging the CRP across the 10 trials (full STS and all 4 periods). Next, the mean of the ensemble curve provided a MARP value for each segment, across each period, effectively describing coordination between two adjacent joints.

4.3.4.2. Inter-joint coordinative variability: Deviation Phase (DP)

To determine within-subject inter-joint coordination variability during the STS task, the deviation phase (DP) was calculated (24). This value describes the magnitude in variation of inter-joint coordination (i.e., in-phase/out-of-phase coupling relationships) for both LHip-L3S1 and L3S1-T12L3 segments across the 10 trials. For each participant, the standard deviation of each point of the 10 CRP trials was calculated across the movement profile (full trial and all 4 periods). The mean of these values was used as DP for each participant. A custom Matlab 2016Rb script (The MathWorks Inc., Natick, MA, USA) script was used for all calculations.

4.3.5. Statistical analysis

Most of the DP and MARP data violated the assumption of normality, based on the Shapiro-Wilkes test. Descriptive statistics were therefore calculated for each variable, following which a $\log_{10}$ transformation was applied to all data, allowing them to meet all criteria for normality. Separate analyses were then completed for the log-transformed dependent variables DP and MARP, at both LHip-L3S1 and L3S1-T12L3. Homogeneity of variance across conditions was measured by Levene's test. Independent t-tests investigated differences between groups across the entire STS task. Two-way mixed design ANOVA, with repeated measures, were run to calculate the effects of group (LBP/Healthy) and period (Start/Up/Down/End), and their interaction. In the event of a violation of the assumption of sphericity, a Greenhouse-Geisser correction was used. Level of significance for all tests was set at $\alpha \leq 0.05$, and a Bonferroni correction was applied for post-hoc pairwise comparisons. All statistical analyses were performed using SPSS for Windows Version 23 (IBM, Chicago, IL, USA).

Table 4.1. Means (SD) for subject demographic information

	Healthy Group (n=21, 10 females)	**LBP Group** (n=16, 11 females)
Age (y)	27 (10)	30 (9)
Weight (kg)	67.4 (10.0)	**78.6 (18.5)**
Onset of symptoms (months)	-	109.9 (113.5)
NPRS (/10)	-	3.4 (1.1)
ODI (%)	-	25.3 (7.4)
Start Back Screening Tool Score	-	4.4 (1.8)

NPRS: Numerical pain rating scale; ODI: Oswestry Disability

Bolded value represents significant difference ($P < 0.05$)

4.4. Results

Means and standard deviations for inter-joint coordinative variability (DP) and inter-joint coordination (MARP), for LHip-L3S1 and L3S1-T12L3, are presented in Table 4.2

4.4.1. Inter-joint coordinative variability (DP)

For LHip-L3S1, the LBP group showed significantly more variability in inter-joint coordination (DP) over the full STS movement ($P = 0.013$; mean difference = -6.95, 95% CI = -12.3 to -1.59) (Figure 4.1A). When examining separate STS periods, significant group, period, and group-period interaction effects were also found (Table 4.3), with pairwise comparisons indicating more variability in inter-joint coordination for the LBP group during the start period ($P < 0.001$; mean difference = -0.325, 95% CI = -0.493 to -0.156) (Figure 4.1B).

For L3S1-T12L3, no between-group difference in DP was found over the full STS movement ($P = 0.103$). For the STS periods, however, there was a significant period effect, characterized by greater variability over the start and end periods of the STS task, with no group or interaction effects (Table 4.3).

4.4.2. Inter-joint coordination amplitude (MARP)

For LHip-L3S1, the LBP group showed more out-of-phase inter-joint coordination (higher MARP) over the full STS movement ($P = 0.010$; mean difference = -22.6, 95% CI = -39.1 to -6.03) (Figure 4.2A). For the STS periods, significant group, period, and group-period interaction effects were found (Table 4.3), with pairwise comparisons showing higher MARP for the LBP group during the start ($P < 0.01$; mean difference = -0.350, 95% CI = -0.549 to -0.150) and end periods ($P < 0.01$; mean difference = -0.354, 95% CI = -0.602 to -0.105) (Figure 4.2B).

For L3S1-T12L3, the LBP group also showed more out-of-phase inter-joint coordination (higher MARP) over the full STS movement (P = 0.015; mean difference = -21.0, 95% CI = -37.6 to -4.38). For the STS periods, significant group and period effects were found, but with no interaction effect (Table 4.3). The LBP group had greater out-of-phase inter-joint coordination behavior for all periods, while both groups had more out-of-phase patterns during the start and end periods.

Table 4.2. Mean (SD) values across LBP and healthy groups for coordination (MARP) and variability (DP) in degrees.

Joint Pair	Period	LBP		Healthy	
		DP (°)	MARP (°)	DP (°)	MARP (°)
LHip-L3S1	*Start*	19.20 (9.28)	48.78 (29.67)	12.25 (5.20)	26.20 (12.57)
	Up	19.17 (9.34)	54.07 (37.44)	9.88 (7.45)	22.77 (16.61)
	Down	11.68 (9.81)	31.54 (37.30)	9.36 (4.57)	19.97 (7.92)
	End	12.20 (10.01)	38.77 (25.74)	8.97 (4.71)	26.57 (13.60)
	Full	18.99 (13.0)	59.27 (40.39)	12.77 (7.52)	26.54 (22.69)
L3S1-T12L3	*Start*	24.40 (8.22)	62.83 (28.18)	20.46 (6.07)	41.85 (17.78)
	Up	22.29 (10.33)	60.87 (44.31)	16.26 (10.17)	29.07 (22.43)
	Down	14.69 (8.35)	43.22 (38.37)	14.44 (7.08)	28.98 (13.25)
	End	21.34 (10.35)	53.55 (24.27)	18.02 (7.68)	51.32 (27.54)
	Full	23.52 (14.58)	67.90 (40.64)	17.19 (7.84)	39.09 (29.49)

Table 4.3. Summary of ANOVA results for log-transformed deviation phase (DP) and mean absolute relative phase (MARP).

	DP		MARP	
Joint Pair	LHip-L3S1	L3S1-T12L3	LHip-L3S1	L3S1-T12L3
	F-test (p-value)			
Main Effect				
Group	5.26 (**0.028)**	2.20 (0.147)	7.96 (**0.008)**	5.91 (**0.020)**
Period	6.43 (**0.003)**	3.59 (**0.022)**	4.76 (**0.004)**	5.78 (**0.001)**
Interactions				
*Group*Period*	3.96 (**0.023)**	1.31 (0.278)	4.46 **(0.005)**	2.13 (0.101)

Significant differences ($P < 0.05$) are in bold

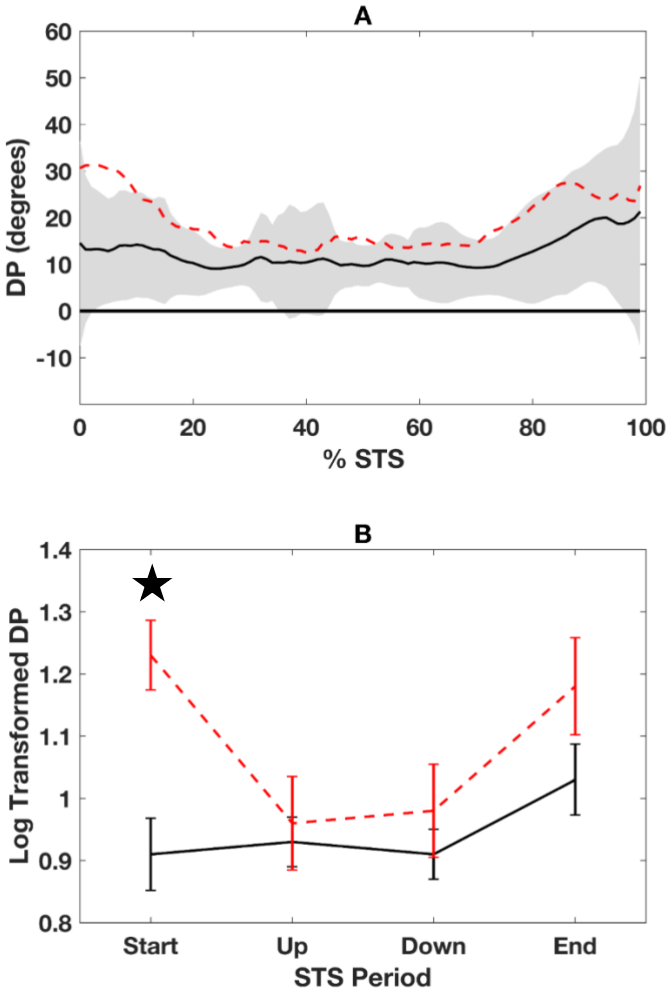

Figure 4.1. Inter-joint coordination variability/deviation phase (DP) across the full task and individual periods in low back pain and healthy groups: Ensemble group means of coordinative variability (DP) across the full sit-to-stand-to-sit (STS) task (A) and mean log transformed DP across each STS period (B) for the LHip-L3S1 segment in adults with LBP (red dashed line) and healthy individuals (solid black line). The shaded area (A) represents the standard deviation for healthy subjects, and error bars (B) are the standard error for both groups. The star denotes a significant difference ($P < 0.05$).

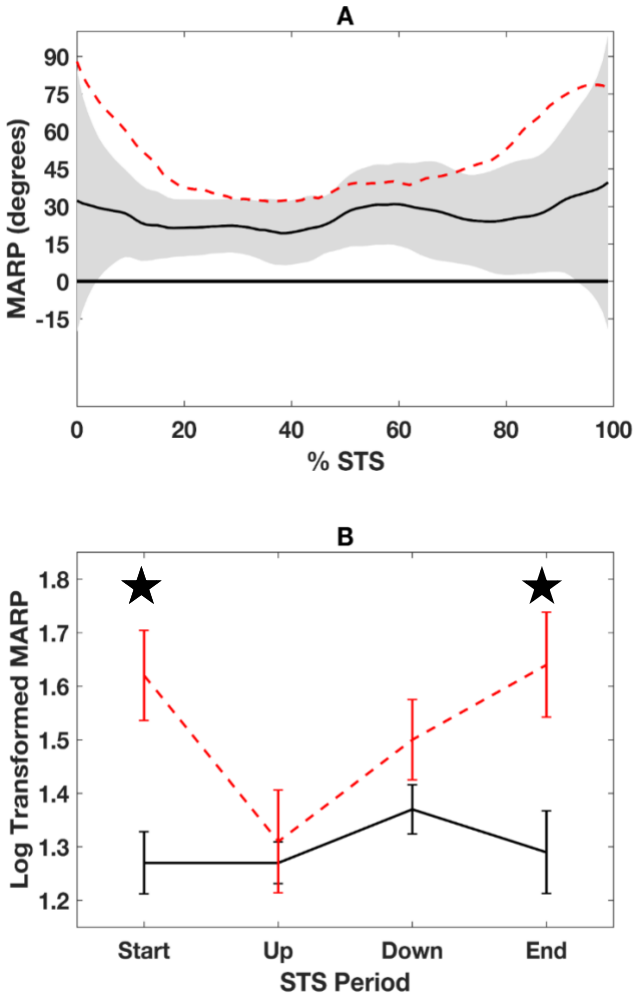

Figure 4.2. Inter-joint coordination/mean absolute relative phase (MARP) across the full task and individual periods in low back pain and healthy groups: Ensemble group means of inter-joint coordination (MARP) across the full sit-to-stand-to-sit (STS) task (A) and mean log transformed MARP across each STS period (B) for the LHip-L3S1 segment in adults with LBP (red dashed line) and healthy individuals (solid black line). The shaded area (A) represents the standard deviation for healthy subjects, and error bars (B) are the standard error for both groups. The star denotes a significant difference ($P < 0.05$).

4.5. Discussion

Contrary to our expectations, our data indicate that the LBP group had more variable (higher DP) and more out-of-phase (higher MARP) patterns of inter-joint coordination in the lumbo-pelvic region, particularly at the beginning and end of the STS task. This indicates clear differences in motor coordination between groups, which is consistent with past research (5, 8, 9, 25, 26). By assuming that the movement variability exhibited by pain-free individuals represents an "optimal" behavior, we can infer that differences in individuals with LBP represent motor control "dysfunction". Such an inference is reasonable, to a point, but must be made with caution and while considering more than just physical factors (27).

Greater variability in inter-joint coordination (higher DP) between the LHip and L3S1 segments (LHip-L3S1) in the LBP group was most evident in the start phase of the STS task (Figure 4.1A). This suggests that the nature of the task must be considered when investigating movement variability in this population. Increased coordinative variability has previously been observed in people with chronic LBP during a reaching task (8), while reduced variability (lower DP) of coordination patterns have been reported in this patient population during running (25) and bending (5) – although the latter was only observed when task demands were increased. To further complicate matters, Lamoth et al. (2006) reported both increased and decreased lumbo-pelvic movement variability in chronic LBP during gait, in the frontal and transverse planes, respectively. Such inconsistencies across studies, may relate not only to the task conditions, but also to differences in sample characteristics and methodology relating to CRP calculations (22, 28).

It has recently been proposed that LBP-related changes in motor control are based on individuals learning to adopt specific movement strategies aimed at minimizing pain or the threat of pain, leading to reduced motor variability (in terms of trunk kinematics and muscle recruitment)

(29). Our findings of excessive variability in the lumbo-pelvic complex, however, are not in agreement with this. One possible interpretation is that the STS task in our study was not threatening enough to elicit a protective stiffening motor strategy in our LBP group, as may be inferred from our sample's low psychological distress associated with their back pain (Table 4.1). As such, it is possible that only key goal-related performance variables (e.g. center of mass movement (30)) were tightly controlled, and that other elements (i.e. exact lumbo-pelvic joint configurations) were left free to vary (30). Such movement variability is functionally necessary, as it allows for many motor solutions to a particular task (2, 31). Alternatively, when considering past reports of impaired proprioception in LBP (32), excessive movement variability may be interpreted as the result of imprecise sensory feedback and/or motor commands in the spinal system. Such imprecisions could lead to greater deviations away from the intended movement trajectory and less accuracy in any required corrections (10), contributing to an increase in movement variability. The surplus of movement variability observed in the current study was most evident when initiating the movement to stand (i.e., start period), while transferring weight to the feet. This requires significant lumbo-pelvic flexion (12), and was performed quickly, which represents a substantial challenge to the system. During such a challenge, imprecisions in sensory feedback and/or motor commands may become more apparent, creating more movement variability, and rendering the system less adaptable in the event of a perturbation (3). Our LBP group, however, had relatively low levels of pain (NPRS) and pain-related disability (ODI), and completed the task without symptom exacerbation. While a clear difference in motor control was observed, classifying it as dysfunctional behavior may not be fully warranted.

Our results also indicated greater out of phase inter-joint coordination (higher MARP) in the LBP group, for both LHip-L3S1 and L3S1-T12L3, across the full task (Figure 4.2A), and

particularly during the start and end periods for LHip-L3S1 (Figure 4.2B). Similar to our findings for coordinative variability (DP), the effect of period on inter-joint coordination indicated more out-of-phase behavior (higher MARP) during the start/end periods for both groups of participants. A previous investigation of lumbo-pelvic coordination during STS in sub-acute LBP reported somewhat different findings: more in-phase initially (i.e., start) and more out-of-phase during the standing/sitting (i.e., up and down) motion (16). It should be noted, however, that their sample included participants with and without sciatica, and with higher pain intensity. The difference may, therefore, be indicative of a direct influence of pain on movement coordination, perhaps to redistribute spinal load across injured tissues (33), or on spinal proprioception (32). This previous study, however, also considered the lumbar spine as a single unit, and used different methods for task-period event identification. Other work, however, also points toward greater in-phase coordinative patterns (lower MARP) during movement in chronic LBP (5, 25, 26), while one study investigating reaching found more out-of-phase lumbo-pelvic coordination (higher MARP) in individuals with chronic LBP (8). Situating our findings in the greater body of research, therefore, may speak to differences in task complexity, or to the heterogeneous nature of LBP.

Our findings should be interpreted in light of certain study limitations. First, the cross-sectional nature of this study limits the causal inference on the above-mentioned associations. Further, in the context of the biopsychosocial nature of LBP, cognitive factors such as fear and catastrophizing have been shown to relate to physical factors in this population (34, 35). While we did collect data related to physical and psychosocial prognostic factors (i.e., STarT Back scores), perhaps additional tools measuring the above-mentioned constructs could have made for a more complete discussion of observed motor behavior. Additionally, our LBP group was also significantly heavier than our healthy subjects; thus, we cannot disregard the possibility that some

of our observations may relate to differences in body mass between groups. Finally, it is possible that our sample was not reflective of a LBP population as a whole, and that different behaviors might have been observed had our sample shown greater levels of pain and disability.

4.6. Conclusion

Our study demonstrated more variable (higher DP) and more out-of-phase (higher MARP) patterns of lumbo-pelvic coordination, particularly during the start and end periods of a STS task, in adults with LBP. While a significant difference in motor control between groups was observed, when considering our sample characteristics (i.e., low pain/disability, low-med STarT back scores) and lack of symptom exacerbation during the STS task, it may be excessive to categorize these findings as representative of dysfunctional motor control. We must consider, however, that a threshold exists beyond which added movement variability is no longer beneficial, and becomes indicative of error in the motor control system.

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Preface to Chapter 5

When completing the study in chapter 4, we encountered a methodological issue involving our Hilbert-transform CRP procedures. These analyses are prone to edge-effects (i.e., data distortion) which can bias CRP values and complicate the clinical interpretation of coordination and variability data. Therefore, chapter 5 used theoretical and kinematic data from people with LBP performing a sit-to-stand-to-sit task, to compare the effects of data padding approaches to control for distortion during CRP analyses. We outlined a simple approach for managing this issue and make a recommendation for future researchers based on empirical data.

Chapter 5: The effects of data padding techniques on continuous relative phase analysis using the Hilbert transform

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5.1. Abstract

Continuous relative phase (CRP) analysis using the Hilbert-transform is prone to end-effects. The purpose was to investigate the impact of padding techniques (reflection, spline extrapolation, extraneous data, unpadded) on end-effects following Hilbert-transformed CRP calculations, using sinusoidal, non-sinusoidal, and kinematic data from a repeated sit-to-stand-to-sit task in adults with low-back pain (n = 16, mean age = 30 years). CRP angles were determined using a Hilbert-transform of sinusoidal and non-sinusoidal signals with set phase-shifts, and for the left thigh/sacrum segments. Root mean square difference (RMSD) and true error compared test signals with a gold standard, for the start, end, and full periods, for all data. Mean difference 95% bootstrapped confidence intervals were calculated to compare padding techniques using kinematic data. The unpadded approach showed near-negligible error using sinusoidal data across all periods. No approach was clearly superior for non-sinusoidal data. Spline extrapolation showed significantly less RMSD (all periods) when compared with double reflection (full period: mean difference = 2.11, 95% CI = 1.41 to 2.79) and unpadded approaches (full period: mean difference = -15.8, 95% CI = -18.9 to -12.8). Padding sinusoidal data when performing CRP analyses is unnecessary. When extraneous data has not been collected, our findings recommend padding using a spline to minimize data distortion following Hilbert-transformed CRP analyses.

5.2. Background

Continuous relative phase (CRP) is an analysis technique used to study joint coordination and variability in human movement (1). CRP is based in dynamic systems theory and quantifies the phase relationship between two body segments (2). A recent review suggests that the most robust approach of determining CRP is based on a Hilbert-transform (2). However, the Hilbert-transform results in end-effects (i.e. data distortion), similar to the difficulties encountered during digital filtering (3). Such distortions would preclude interpretation of CRP waveforms at the start and end of the movement.

Past work suggests managing Hilbert-transform related end-effects by analyzing a window of reliable data (4), or by extending data beyond its range using padding techniques (4-6). These techniques were described using multi-step methodology (i.e., Hilbert-Huang Transform) and analyzed mechanical vibration or geophysics data. Attention has been paid to manage this issue in engineering research, but this has not been examined using biomechanical data in CRP analysis. Geophysical/vibration data behaves differently (i.e., repeated waveform) than biomechanical, kinematic data (i.e., irregular single movement cycle). Thus, controlling end-effects following Hilbert-transform CRP calculations in biomechanics should be investigated.

Data reflection (7), polynomial extrapolation (8), and recording extraneous data (9), are methods of controlling end-effects from digital filtering of kinematic data. Collecting extraneous data is regarded as common practice; however, if this is not considered during study design or there is a limitation in capture volume, such data may not be available or sufficient in length. Applying the above-mentioned techniques to manage Hilbert-transform induced end-effects seems reasonable, but has yet to be investigated.

One challenge when performing CRP analyses on biomechanical data is the lack of advance knowledge of the "true" phase relationship between two segments. As a result, when studying the methodological aspects of CRP analysis, including series with known relative phases (e.g., sinusoidal waves with set phase shift) is common practice and provides greater insight into the effects of different variables on phase relationships (2). Therefore, the objective is to investigate the impact of three methods of data padding (double reflection, spline extrapolation, no padding) on end-effects following Hilbert-transformed CRP calculations, in comparison with a gold standard, using (i) sinusoidal data, (ii) non-sinusoidal data, and (iii) lumbo-pelvic kinematic data during a sit-to-stand-to-sit task in adults with low back pain. A secondary objective will be to determine the optimal pad length which is most effective in controlling end-effects following Hilbert-transformed CRP calculations, when using double reflection and spline extrapolation techniques on kinematic data. We hypothesize that spline and double reflection padding techniques will result in smaller end-effects than unpadded methods when compared to a "gold standard" using sinusoidal, non-sinusoidal, and empirical (kinematic) data.

5.3. Methods

5.3.1. Sinusoidal and nonsinusoidal data

Consistent with past authors, sinusoidal and non-sinusoidal signals were mathematically generated using a series of equations (2). First, a sinusoidal wave, sampled at 1000 Hz, was generated using equation 1 and compared with an identical signal with a horizontal phase shift of 18° (Figure 5.1A). A non-sinusoidal wave was then produced using equation 2 and compared with an identical signal with a 126° horizontal phase shift (Figure 5.2A).

$$Eq.1: x(t) = \sin(2t), t \hat{I} [0, 2\rho]$$

$$Eq.2: x(t) = \frac{\cos(t - 0.25\rho)}{\sqrt{1 + 0.41418^2 - 2 \acute{} 0.41418\sin(t - 0.25\rho)}}$$

5.3.2. Kinematic data collection and processing

Previously collected data from 16 participants with low-back pain were used (11 females; mean (standard deviation) age: 30 (9) years; body mass: 78.6 (18.5) kg) (10). Subjects were sub-acute and chronic in nature (mean duration of symptoms 109.9 (113.5) months) with pain located between the lower ribs and gluteal folds and no evidence of specific low back pain (e.g., spinal stenosis, vertebral compression fracture) or serious pathology (e.g., cancer). Informed consent was obtained for all participants.

Procedures have been described (10). Briefly, participants completed 10 trials of a sit-to-stand-to-sit task while kinematic data were acquired using an electromagnetic TrakSTAR motion capture system (Ascension Technology, Milton, VT, USA), sampled at 200 Hz. Data from sensors (model 800) applied to the lateral left thigh (LThigh) and base of the sacrum (S1) were used for analyses. A force plate (BP400600 NC, AMTI, Watertown, MA, USA) was mounted on the seat to capture surface reaction forces under the thighs and buttocks, sampled at 200 Hz, in order to identify sit-to-stand-to-sit events.

Force plate and kinematic data were smoothed using a low-pass, fourth-order, bi-directional Butterworth filter, with a cut-off frequency of 10 Hz. Orientation data were used to determine segment angles in the sagittal plane for LThigh and S1 segments, relative to the lab. Our area of interest was the “up/down” phase of the sit-to-stand-to-sit waveform. This sub-section was defined as the point of loss of contact with the seat as indicated by a vertical reaction force of < 10 N (standing up), to the point of contact with the seat on the return, as measured by the initial

recording of a vertical reaction force > 10 N (sitting down). Data from the full waveform before and after the "up/down" phase were retained and used as extraneous data to pad our criterion signal.

5.3.3. Padding methods

For the sinusoidal and non-sinusoidal waves, three different approaches padded our signals with 100 additional data points: double reflection, spline extrapolation, and extraneous data. A fourth, unpadded approach (no extra data was added) was included to illustrate end-effects. For these waves, we retained the entirety of the signal. A padding length of 100 data points was selected by calculating 10% of the sampling frequency (1000 Hz). Double reflection (7) involves reflecting a set amount of data points around an endpoint, effectively flipping it about the horizontal and vertical axes. This was performed at the start and end of our waveform. Cubic spline extrapolation was also used (8), effectively padding our signal with 100 data points on either end. To collect extraneous data, our waveforms were extended by having our signals repeat across three time periods and 100 additional data points were retained prior to, and at the end, of our original signal. For our investigation, all padding and unpadded methods are referred to as test signals.

To assess the impact of padding approaches on end-effects, a test signal for each padding method was compared with a gold standard (i.e., criterion signal). For our sinusoidal waves, given the signals have the same frequency, we would expect them to behave identically and demonstrate a constant continuous relative phase equal to 18°. Any deviations from this value would be considered to represent frequency artifacts (11). As such, our criterion signal will be a constant relative phase value of 18° for the entirety of the waveform. For our non-sinusoidal signals, although they have the same frequency, the irregular nature of the waveform (i.e., non-sinusoidal)

dictates that the two signals will be constantly increasing and decreasing their phase shift of 126° while never being fully in-phase with one another (2). Thus, for our non-sinusoidal data we would expect the CRP to oscillate around 126° for the duration of its cycle. This value will represent our criterion signal for the non-sinusoidal waveform.

Likewise, three approaches were used to pad our kinematic signals from the sit-stand-to-sit-task with 100 data points at the start and end of our up/down waveform. As described above, the double reflection, spline extrapolation, and unpadded techniques were employed. In this case, the gold standard (i.e., criterion signal) was defined as the aforementioned up/down phase, with 100 additional real data points (i.e., extraneous data from entire sit-to-stand-to-sit waveform) retained both before and after the up/down phase (Figure 5.3). These methods were performed for S1 and LThigh segments and repeated across each of the 10 trials, for all 16 subjects.

5.3.4. Continuous relative phase

For the sinusoidal and non-sinusoidal data, a Hilbert-transform was applied to the test signals and phase angles across the two waveforms were determined (2). Consistent with recommendations by Lamb and Stöckl (2012), our data were amplitude-centered around zero (prior to data padding) and no normalization techniques were used. The absolute difference in phase angles between waveforms across the entire cycle quantified the CRP for the two signals. Data pads were removed (when appropriate) and the CRP waveform corresponding to the original cycle was retained. This procedure was repeated for each padding method (double reflection, spline extrapolation, extraneous data) and the unpadded approach.

Similar procedures were used to analyze kinematic data. A Hilbert transform determined phase angles for the S1 and LThigh segments across the entire trial and quantified the CRP for the

LThigh-S1 joint pair. Data pads were removed (when appropriate) and the CRP waveform corresponding to the up/down phase for LThigh-S1 was retained. This procedure was repeated for three padding methods (double reflection, spline extrapolation, unpadded) and the criterion signal, across each trial, for all 16 subjects. All data processing was performed using custom scripts in MATLAB 2016Rb (The MathWorks Inc., Natick, MA, USA).

5.3.5. Pad length

To determine the optimal length for an extrapolation window, a simulation testing varying lengths of data pads was run using collected kinematic data for the Lthigh and S1 segments, for each subject. A representative trial corresponding to the up/down waveform was retained and incrementally padded with 10 points, on either end, up to a limit of 500 additional points (i.e., 10, 20, 30, … 500). For each iteration, as described above, Hilbert-transformed CRP analyses were performed for the Lthigh-S1 joint pair and compared with a gold standard (identical signals padded with 100 points of extraneous data prior to CRP analyses). This was repeated for each participant and for the double reflection and spline extrapolation techniques.

5.3.6. Statistical analysis

Descriptive statistics were calculated for all variables using SPSS version 20.0 (IBM). To quantify inconsistencies between the criterion and each test signal, root mean square difference (RMSD) and true error were calculated. True error was defined as our test signal subtracted from the criterion signal. A negative value indicated underestimation of the test signal relative to the criterion signal, while a positive value was an overestimation. For all data (i.e., sinusoidal, non-sinusoidal, and kinematic), end-effects were assessed using mean RMSD and true error values for

the first and last 10% of our signal (start and end periods). The same calculations were also performed for the entire retained waveform (full period). For the kinematic data, these calculations were performed for each trial and averaged over the 10 trials for each participant.

To provide meaningful comparisons between padding approaches (e.g., spline extrapolation vs. no padding) when analyzing our kinematic data, mean difference 95% bootstrapped confidence intervals were calculated for RMSD and true error, using the percentile method (12). This was done across the start, end, and full periods. Participant data were resampled 1000 times. For each iteration, differences between padding approach RMSD and true error means were calculated. Based on these 1000 iterations, 95% confidence intervals were determined. If the 95% confidence intervals did not include 0, we concluded the approaches were significantly different.

To determine the optimal length of data pads, RMSD was calculated between the padded test signal and the gold standard. This was performed for each iteration of pad length, for both padding techniques, and averaged across the 16 participants. The pad length corresponding to the smallest RMSD will represent the optimal window of extrapolation for the respective approaches.

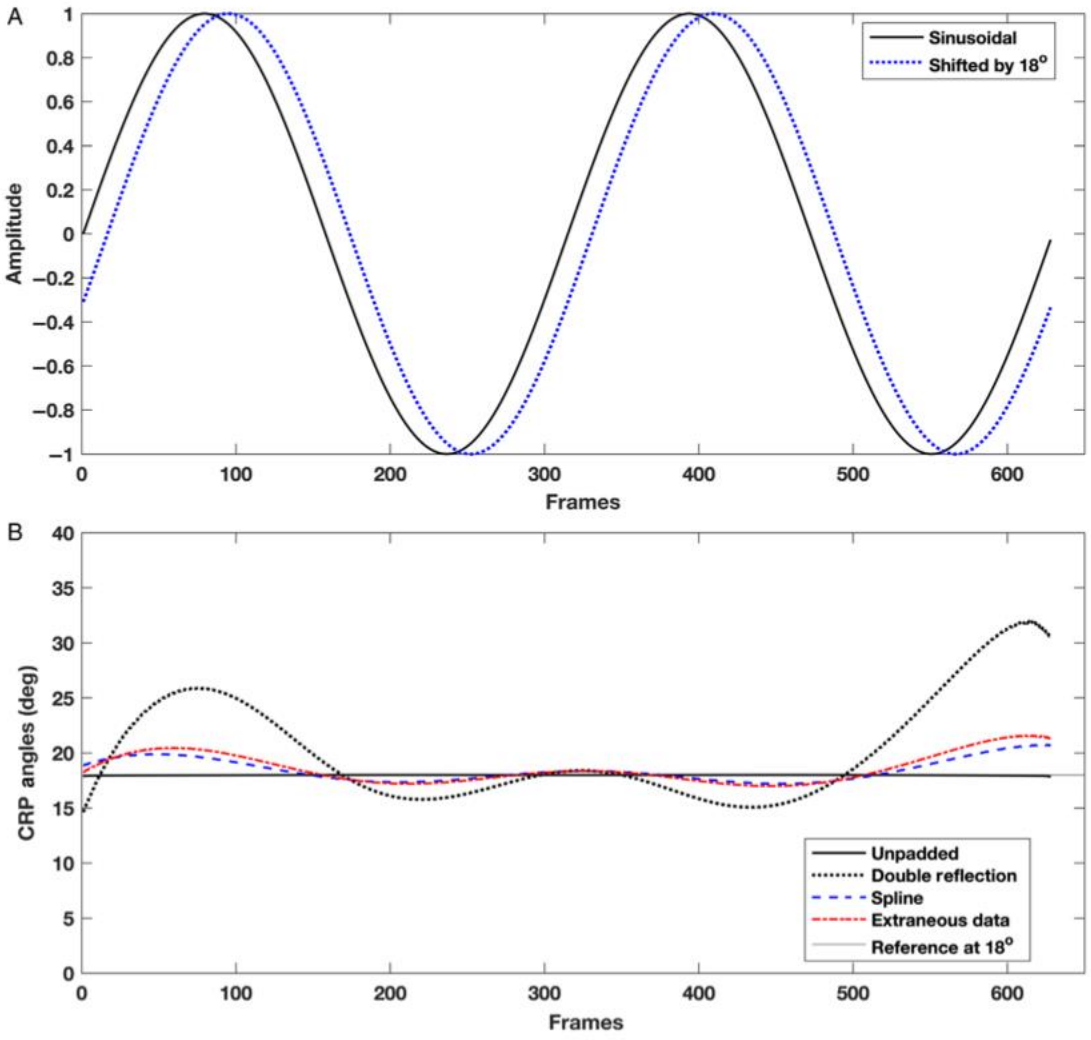

Figure 5.1. Sinusoidal signal shifted by 18°: Two sinusoidal signals shifted by 18° (A) and their corresponding Hilbert-transformed CRP angles following various padding approaches (B). The horizontal reference line represents the expected phase relationship and overlaps with the unpadded approach. CRP indicates continuous relative phase.

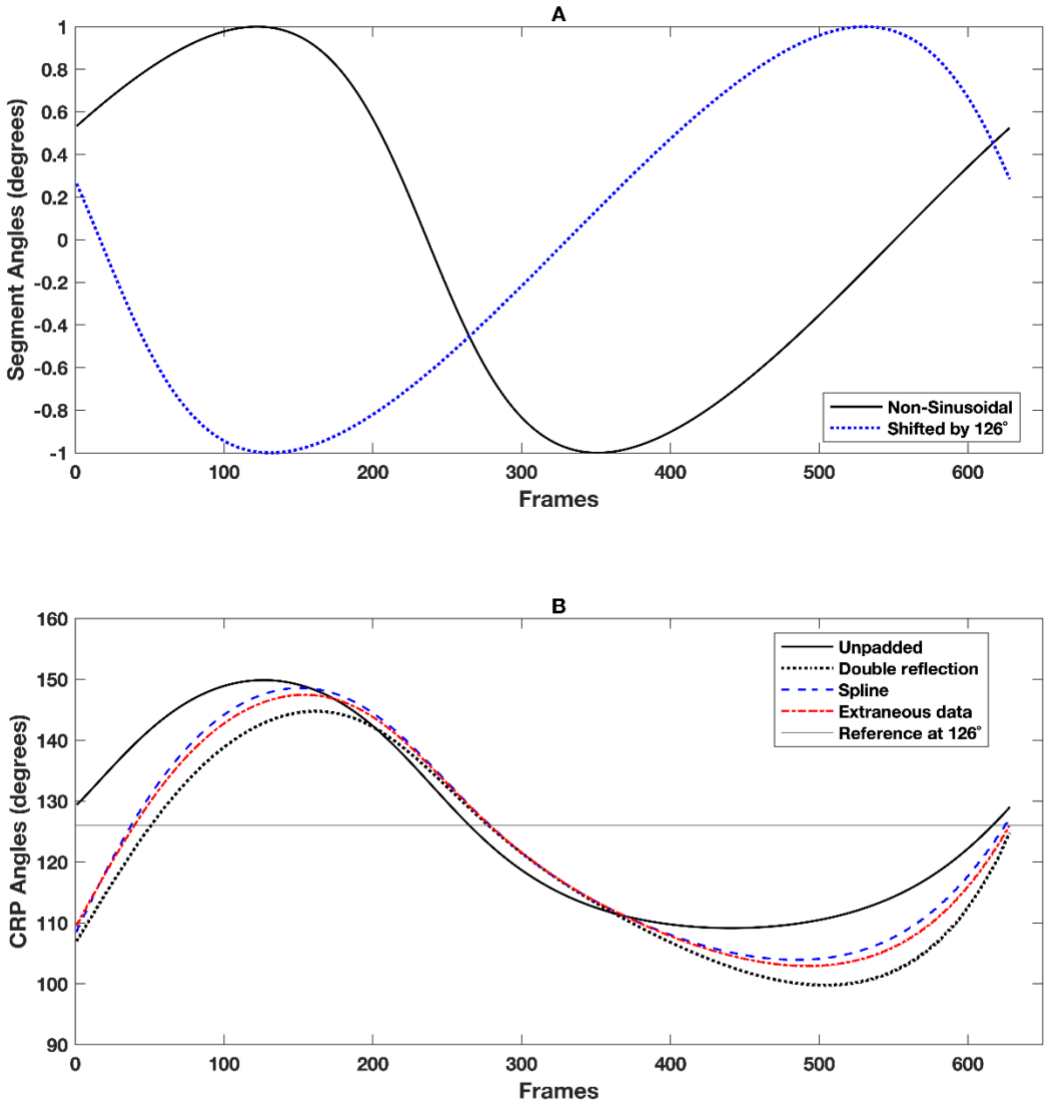

Figure 5.2. Nonsinusoidal signals shifted by 126°: Two nonsinusoidal signals shifted by 126° (A) and their corresponding Hilbert-transformed CRP angles following various padding approaches (B). The horizontal reference line represents the value around which this phase relationship is expected to fluctuate. CRP indicates continuous relative phase.

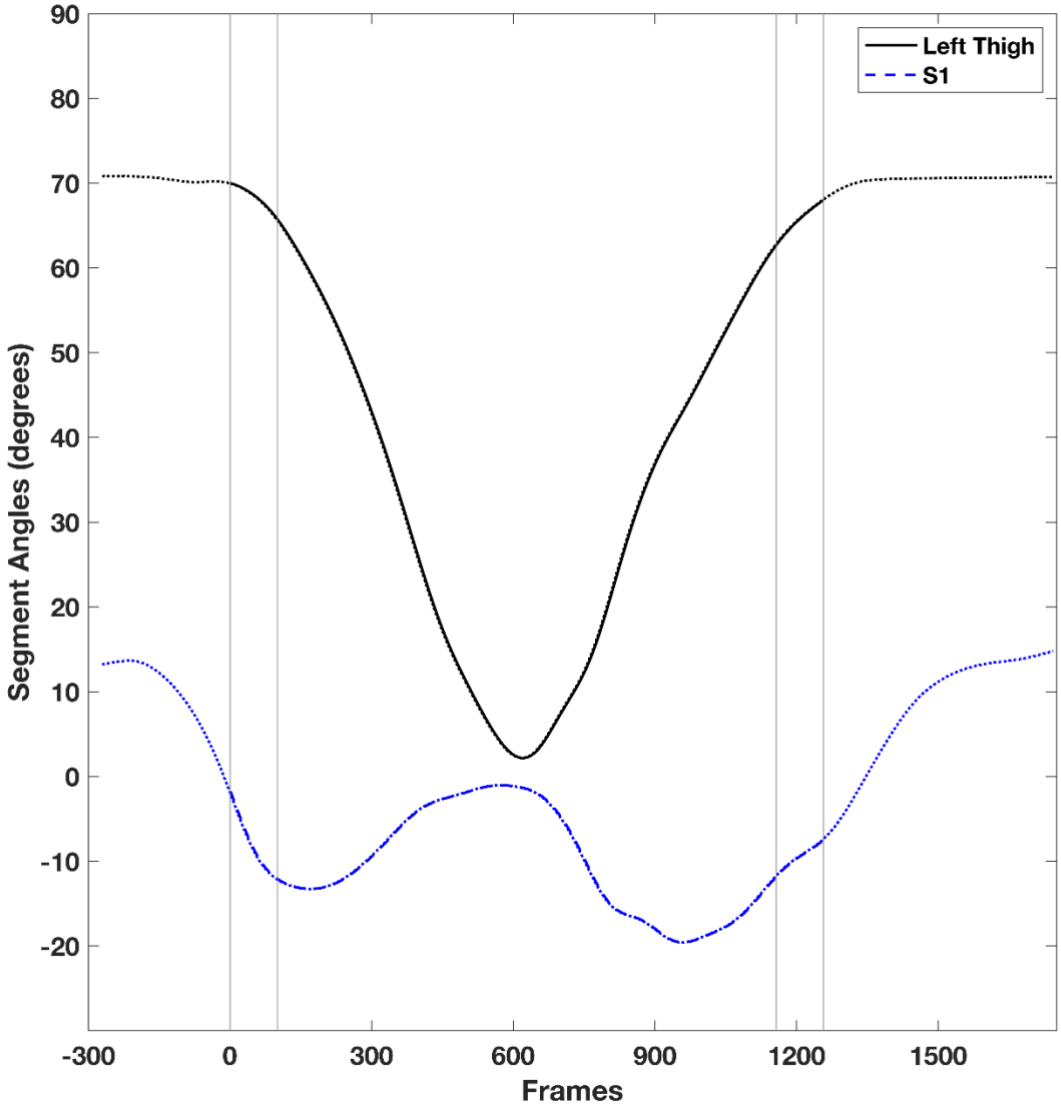

Figure 5.3. Segment angles of the left thigh and S1 during a representative sit-to-stand-to-sit trial: Vertical lines delineate data pads (100 points) at the outset and after the waveform for the up/down phase (middle region). Dotted lines beyond data pads denote additional extraneous data collected and correspond to the entire sit-to-stand-to-sit task.

5.4. Results

Continuous relative phase analysis of two sinusoidal waves with a known phase shift of 18 degrees showed the unpadded approach to be most effective at controlling signal distortion (in terms of both RMSD and true error), across all periods (Table 5.1). These findings suggest that any form of padding, double reflection in particular, will result in end-effects and a degree of distortion across the entire waveform of a repeated sinusoidal signal. On average, signal distortion was in the positive direction (i.e., over-estimation of error).

Analysis of two non-sinusoidal waves with a known phase shift of 126 degrees showed no padding technique to be clearly superior at controlling end-effects (Table 5.1). On average, in terms of both RMSD and true error, the unpadded approach generated the greatest degree of waveform distortion in the start period but produced the smallest error in the full and end periods. While spline extrapolation and extraneous data padding approaches performed similarly with less distortion in the start period (i.e., first 10% of the signal), the spline technique showed slightly less error in the end and full periods. Double reflection generated the greatest degree of distortion in the same periods (end, full).

Using kinematic data from a sit-to-stand-to-sit task, the unpadded approach showed the highest RMSD and true error (in the positive direction) across all periods (full, start, end) when comparing criterion and test signals following CRP calculations (Table 5.2). This highlights the magnitude of signal distortion that occurs when end-effects are not controlled for and suggests that CRP values tend to be inflated. Spline extrapolation padding generated the smallest RMSD and true error values (least error) for all periods (Table 5.2).

Mean difference 95% bootstrapped confidence intervals of RMSD and true error values showed the unpadded approach resulted in significantly greater RMSD (more error) across all

periods (start, end, full) when compared with the two padding techniques (Table 5.3). This suggest that both double reflection and spline extrapolation are effective in controlling end-effects. Additionally, the unpadded approach showed significantly greater true error (in the positive direction) when compared with double reflection and spline extrapolation, in the full, start, and end periods, respectively. This suggests that foregoing data padding inflates CRP values not only at the ends, but also across the entire signal, relative to the other techniques (Figure 5.4). When comparing spline extrapolation and double reflection, the spline extrapolation technique resulted in significantly smaller RMSD and true error for all periods.

The pad lengths most effective at controlling end-effects in the kinematic data for the sit-to-stand task following Hilbert-transformed CRP analyses corresponded to a length equal to 10 % and 12 % of the sampling frequency (i.e., 100 and 120 points) for the spline extrapolation (RMSD = 1.03, standard error = [-0.13, 0.13]) and double reflection (RMSD = 2.28, standard error = [-0.20, 0.20] techniques, respectively (Figure 5.5). Beyond these inflection points, a near-linear rise in error is observed for both techniques as the pad length is increased, most notably with the spline approach. Overall, the spline extrapolation method with a pad length of 100 points (10% of sampling frequency) generated smaller RMSD and standard error values than the double reflection approach.

Table 5.1. Root means square difference (RMSD) and true error between test and criterion signals for sinusoidal and non-sinusoidal data following continuous relative phase analysis.

		RMSD (º)			**True Error (º)**		
	Padding technique	*Start*	*End*	*Full*	*Start*	*End*	*Full*
Sinusoidal wave	*Unpadded*	0.04	0.05	0.02	-0.03	-0.04	-0.01
	Double reflection	4.86	12.4	5.24	3.62	12.3	2.25
	Spline	1.65	2.30	1.08	1.63	2.27	0.42
	Extraneous	1.91	3.19	1.45	1.80	3.17	0.60
Non-sinusoidal wave	*Unpadded*	12.1	5.60	14.9	11.2	-4.13	0.02
	Double reflection	9.71	14.8	16.7	-7.00	-13.6	-6.15
	Spline	8.40	10.1	15.9	-2.97	-8.65	-3.14
	Extraneous	7.99	11.6	16.0	-3.43	-10.3	-3.90

Table 5.2. Mean (SD) values for root mean square difference (RMSD) and true error between test and criterion signals following continuous relative phase calculations for kinematic data from the sit-to-stand-to-sit task.

Padding Technique	**Period**	**RMSD** (º)	**True error** (º)
Unpadded	*Full*	16.6 (6.68)	8.40 (4.28)
	Start	25.6 (11.8)	13.5 (16.7)
	End	19.5 (8.85)	15.8 (9.11)
Double Reflection	*Full*	2.85 (1.55)	1.19 (0.86)
	Start	5.56 (4.01)	3.00 (3.92)
	End	4.14 (3.38)	3.24 (3.12)
Spline Extrapolation	*Full*	0.76 (0.35)	-0.03 (0.16)
	Start	1.02 (0.90)	-0.42 (0.58)
	End	0.64 (0.44)	0.26 (0.43)

Table 5.3. Summary of root mean square difference (RMSD) and true error mean differences [95% confidence interval] between data padding methods for kinematic data from the sit-to-stand-to-sit task.

Error	Period	Mean Difference [95% confidence interval]		
		Double Reflection vs. Spline	Double Reflection vs. Unpadded	Spline vs. Unpadded
RMSD	*Full*	2.11 [1.41, 2.79] *	-13.7 [-17.1, -10.6] *	-15.8 [-18.9, -12.8] *
	Start	4.59 [3.08, 6.34] *	-20.1 [-25.6, -14.3] *	-24.7 [-30.2, -18.8] *
	End	3.53 [2.10, 5.03] *	-15.4 [-18.5, -12.4] *	-18.9 [-23.0, -14.9] *
True error	*Full*	1.24 [0.76, 1.71] *	-7.20 [-9.29, -5.07] *	-8.43 [-10.4, -6.33] *
	Start	3.47 [1.55, 5.54] *	-10.6 [-17.5, -3.81] *	-14.1 [-21.4, -6.31] *
	End	3.03 [1.47, 4.77] *	-12.6 [-15.9, -9.41] *	-15.6 [-19.8, -11.3] *

Asterisk denotes significant difference.

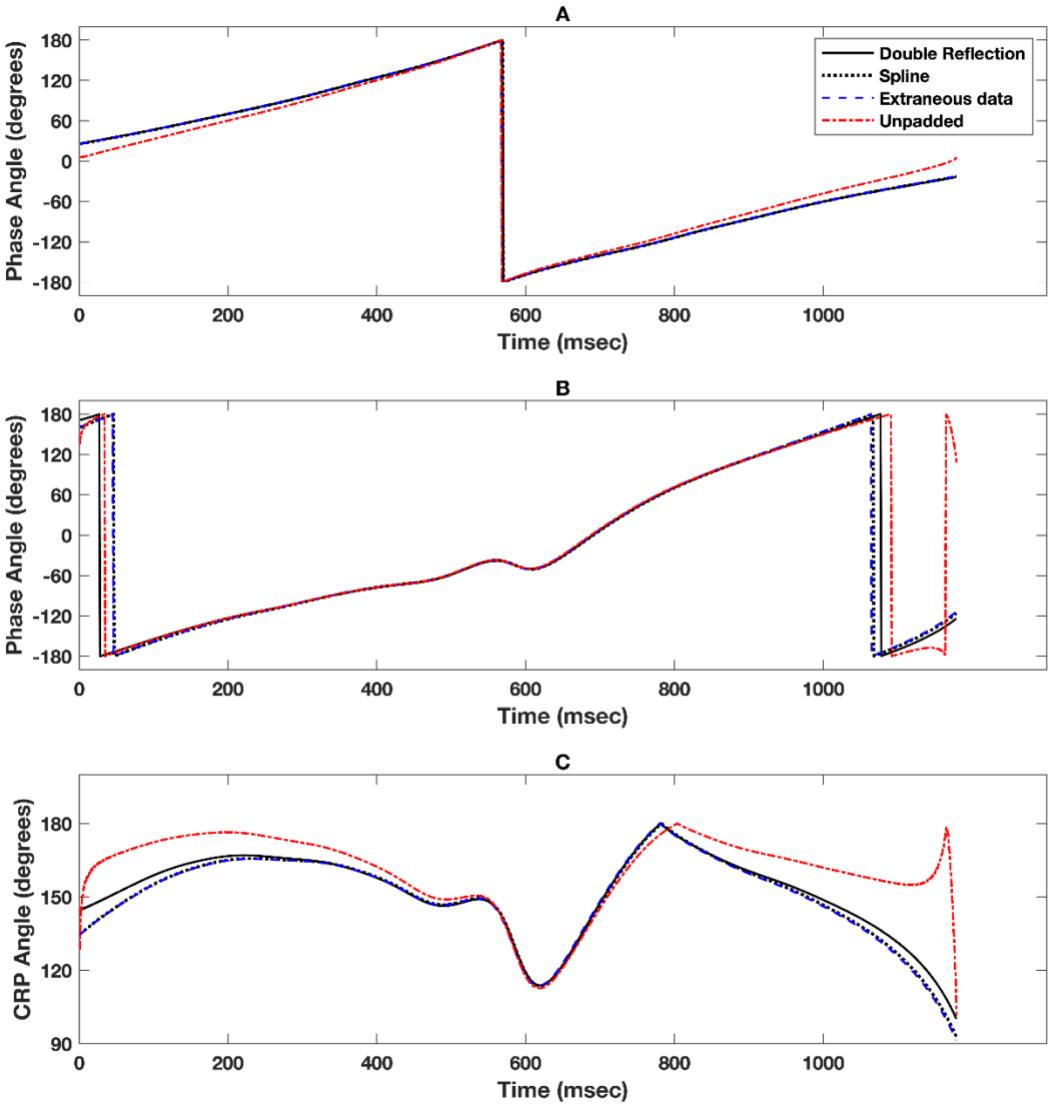

Figure 5.4. A comparison of data padding techniques: A comparison of data padding techniques following Hilbert-transformed CRP analysis: left thigh phase angles (A), S1 phase angles (B), and left thigh–S1 joint pair CRP angles (C). CRP indicates continuous relative phase.

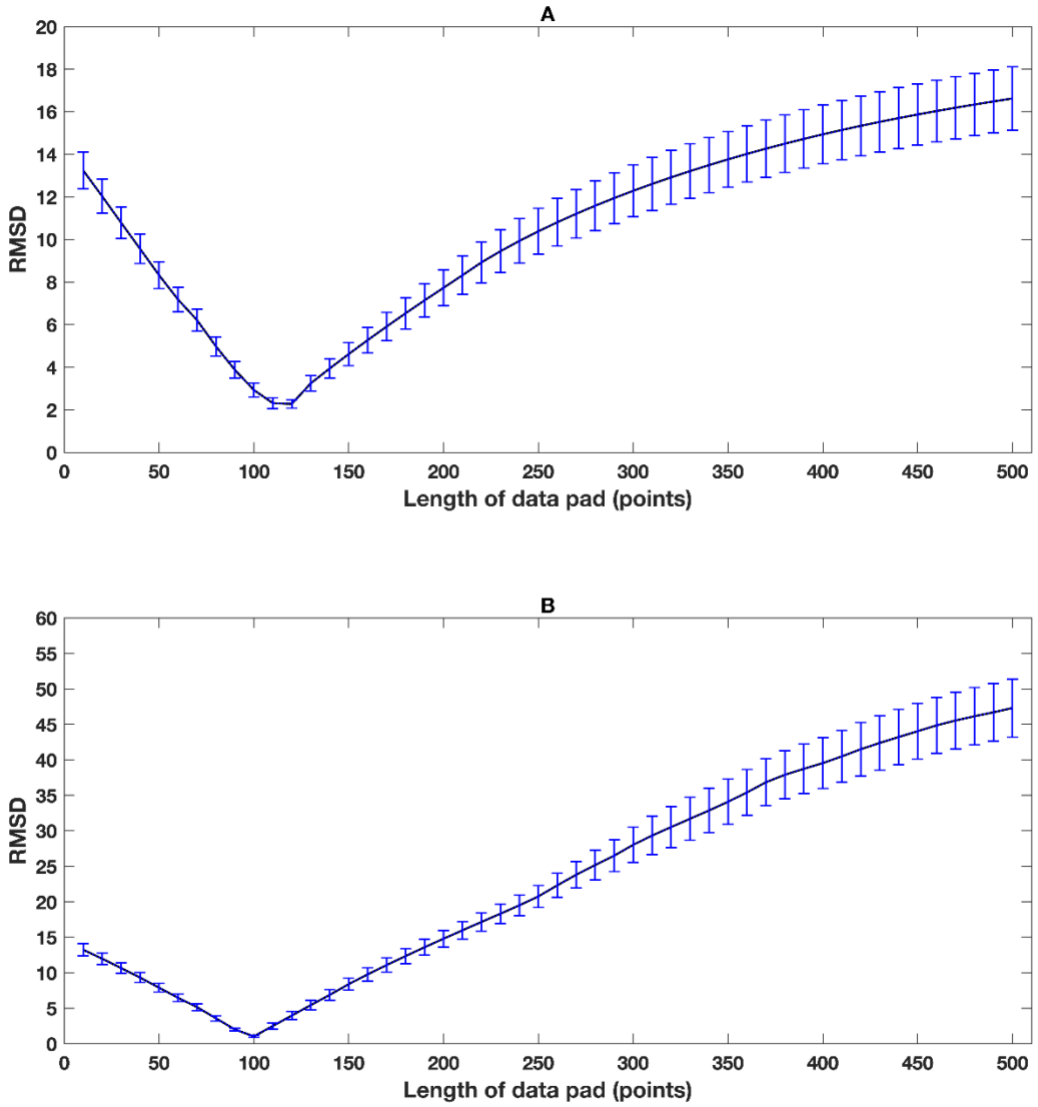

Figure 5.5. Root Mean Squared Difference of continuous relative phase curves and pad length: Average RMSD of continuous relative-phase angles following a padding simulation comparing double reflection (A) and spline extrapolation (B) approaches with extraneous data, using kinematic data from a sit-to-stand-to-sit task. Error bars are standard error for each padding iteration across the 16 participants. RMSD indicates root mean square difference.

5.5. Discussion

There were differences in signal distortion and end-effects when comparing data padding approaches on Hilbert-transformed CRP calculations using sinusoidal, non-sinusoidal, and kinematic data. Our findings suggest that when performing analyses on two sinusoidal waves of identical frequencies, additional padding is unnecessary. These data show that any form of padding produces a greater degree of distortion across all periods (start, end, full) than an unpadded approach. Considering the identical characteristics of the two signals, this finding is somewhat intuitive as attempting to "predict" subsequent data via padding will inevitably change the phase relationship between the two waves. Such waveforms are not commonly found in biomechanical analysis of human movement; thus, these findings are only theoretical in nature. Regarding non-sinusoidal data, our observations of the unpadded approach generating both the most (start period) and least (end, full periods) error are somewhat logical when examining the relationship between the two waves. At the outset, the two waves demonstrate different slopes and move in opposite directions (i.e., greater relative phase), while towards the mid-end of the cycle, the two waveforms are rather similar (i.e., smaller, more stable relative phase) (Figure 5.2). Considering this approach had negligible amounts of true error over the full period (-0.02), this approach may, in fact, most closely reflect the true phase relationship between the two signals.

Performing CRP analyses on kinematic data from a sit-to-stand task without data padding led to considerable end-effects and distortion across the full waveform. The implications of these findings are of interest to movement scientists as these waveforms are typical of kinematic data from human movement. End-effects likely occur due to the Hilbert-transform being performed on a finite segment of data (i.e., start/end points only have one neighboring point) (13), while distortion across the full signal may relate to the Hilbert-transform's sensitivity to abrupt changes

in signal amplitude (3), or differences in centering and/or normalizing procedures (2, 14). Overall, spline extrapolation produced the smallest RMSD and true error, across all periods, and was statistically superior to unpadded and double reflection methods – both of which skewed CRP angles to varying degrees. This requires consideration in CRP analyses where different magnitudes of CRP values have meanings, for example, where low CRP values suggests in-phase coupling and high CRP indicates out-of-phase coupling. While error due to digital processing may be unavoidable altogether, our findings indicate that padding a kinematic waveform using spline extrapolation results in the smallest signal distortion. In the event that future work studying kinematic data has failed to collect extraneous data at the outset of the study, we recommend extrapolating data using a spline technique.

Our findings also suggest that a pad length of 100 data points (10% of sampling frequency), using a spline technique, produced the smallest RMSD values following CRP analyses for our kinematic data set. Interestingly, for both padding approaches, increasing the pad length beyond a certain threshold generated a considerable amount of additional error. This finding may be reflective of our "optimal" pad length and criterion signals being identical in length (i.e., 100 points). Pad length has an effect on waveforms following phase analysis using the Hilbert-transform, thus, comparing data pads with a criterion signal of a different length may have produced different findings. This may relate to the fact that the Hilbert transform is a type of frequency domain analysis. Simply extending the data in the time domain is not a solution, rather, it will distort the Hilbert-transformed signal. In turn, padding techniques should replicate the frequency content of the original data; however, it still risks altering certain waveform properties (e.g., phase shifts).

To our knowledge, no other work has investigated this question using kinematic, or mathematically generated data (i.e., sinusoidal and non-sinusoidal signals) following Hilbert-transformed CRP calculations. Past authors have used a similar structure to the current manuscript and compared the effects of differing techniques (e.g. Hilbert-transform vs. phase portraits) and/or frequency modulation using sinusoidal and non-sinusoidal data (2, 14); however, due to differences in methods, direct comparisons with existing literature cannot be made. Past authors performing Hilbert-transformed CRP analyses of kinematic data reported waveform padding using double reflection (15), mirroring of the entire waveform (10), or did not specify if any technique was used (16). Generally, few details are provided, or the underlying rationale for the selected technique is unclear. Wu and colleagues (2009) reported extrapolating geophysical data based on local extrema as effective in controlling Hilbert-transform related end-effects (4). While this technique may be applicable to repeating, kinematic data (e.g., gait cycle); it would not likely be appropriate for discrete non-repeating waveforms such as individual trials of a sit-to-stand-to-sit task. In digital filtering, Howarth & Callaghan (2009) reported padding kinematic displacement data using linear extrapolation as most effective in controlling end-effects, when compared with double reflection and 3rd order polynomial extrapolation techniques (9).

Certain study limitations exist. Skin marker artifacts may lead to measurement error, especially in obese patients for the sit-to-stand kinematic data. Further, we only analyzed a sit-to-stand-to-sit task, and the generalizability of our findings to other functional tasks (e.g., gait) is limited. Similarly, from our non-sinusoidal data, we cannot extrapolate our findings beyond the presented waveform. Lastly, the Hilbert-transform is sensitive to changing signals, which may have influenced our findings.

In typical kinematic data from human movement, spline extrapolation was the most effective padding technique at minimizing waveform distortion following Hilbert-transformed CRP calculations. When extraneous data is not available, we suggest padding the kinematic waveform using a spline extrapolation technique that is 10% of the sampling frequency in length, prior to CRP calculation.

Acknowledgements

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Preface to Chapter 6

Findings from our systematic review (chapter 3) linked greater pain-related threat and guarded motor behavior; however, few studies reported on this relationship from the standpoint of coordination and variability. Therefore, the next step in this thesis was to study the relationship between pain-related threat and coordination.

The study in chapter 6 involved a new sample of 54 participants with chronic LBP. This work collected data regarding spinal kinematics and pain-related threat (i.e., pain-related fear and pain catastrophizing) during a repeated lifting task and studied spinal coordination and variability using CRP analyses. We chose a repeated lifting task because bending is both problematic and often perceived as more threatening by people with LBP. We hypothesized that greater pain-related threat would be associated with more in-phase and less variable coordination (i.e., "tight" control) during lifting.

Chapter 6: The relationship between pain-related threat and coordination in adults with chronic low back pain during a lifting task: a cross-sectional study.

Patrick Ippersiel, Richard Preuss, Shawn M. Robbins

6.1. Abstract

Background: Changes in coordination may reflect guarded movement in people with low back pain. It is unclear if these behaviors are associated with pain-related threat. We aimed to determine if pain catastrophizing and pain-related fear were related to spinal joint coordination and variability, during a lifting task, in people with chronic LBP.

Methods: Participants were adults with chronic LBP (n = 49; 28 females; mean age = 44.1 (10.8) years). Kinematics for the Hip, LowLx (L3-S1), and UpLx (T12-L3) segments were measured using electromagnetic motion capture during 10 weighted lifting trials. Continuous relative phase analysis using the Hilbert transform method determined coordination and variability of the Hip-LowLx, and LowLx-UpLx joint pairs, deconstructed into 2 periods (lifting/replacing). Pain-related threat was measured using the Tampa Scale for Kinesiophobia and the Pain Catastrophizing Scale. Linear regression analyses tested the relationship between pain-related threat and coordination amplitude and variability, for both joint pairs, during lifting and replacing periods.

Findings: For Hip-LowLx coordination amplitude, the base model (sex) was not significant for both crate lifting and replacing periods. Adding pain catastrophizing explained unique variance and improved our model for lifting (R^2 = 0.147, p = 0.026; r^2 change = 0.125, p = 0.013) and replacing (R^2 = 0.136, p = 0.034; r^2 change = 0.099, p = 0.019) periods, suggesting that greater pain catastrophizing was related to more in-phase coordination. Exploratory analyses and t-tests revealed distinct subgroup aligned with phenotypes of “tight” and “loose” control, where “tight” control was characterized by greater catastrophizing and disability ($p < 0.05$).

Interpretation: Greater pain catastrophizing was related to more in-phase Hip-LowLx coordination during lifting and differentiated between "tight" and "loose" control phenotypes. This suggests that catastrophizing and guarded motor behavior are intertwined; however, the latter findings are preliminary should be interpreted cautiously.

6.2. Introduction

Pain may serve as a protective mechanism, prompting guarding of a region that is perceived to be injured or threatened (1). In people with low back pain, this may manifest as changes in inter-joint coordination and variability. Coordination describes coupling relationships between joints during movement (e.g., hip-spine), while variability reflects the extent to which coordination varies, within an individual, across multiple repetitions of a task. While changes in these behaviors may provide insight into pain and injury in LBP, findings in the literature are conflicting (2, 3).

Recent work proposed distinct phenotypes of "tight" and "loose" control in LBP (4). "Tight" control is a motor behavior which may limit movement via trunk co-contractions in response to pain, or threat thereof (5). "Loose" control is the opposite strategy, whereby reduced trunk excitability enables less constrained control of spinal movement, helping reduce spinal load (4). These subgroups might help reconcile inconsistent reports of coordination in LBP. "Tight" control is consistent with a less variable and more in-phase (i.e., tightly coupled), or guarded, coordination strategy observed in LBP (6). In contrast, more variable and out-of-phase coordination is also observed in people with LBP (2) and may reflect "loose", or less robust control of the spine (4). Persistence of these behaviors, however, may increase compressive loading ("tight") or cause excessive tensile strain ("loose"), and contribute to ongoing pain and re-injury (1, 4). Currently, reasons for divergence in presentation and persistence of symptoms remains unclear.

Lifting is perceived as threatening and often cited as a source of pain and re-injury in LBP (7). Biomechanically, this notion is underscored by a slower and stiffer lifting technique in this group (8). Past work reported "tight" trunk-pelvis coordination (i.e., more in-phase and less variable) during lifting in people with a history of LBP, compared to healthy controls (6). Others

reported more out-of-phase spinal coordination (i.e., “loose”) in people with greater LBP-related disability during lifting, relative to a low LBP-related disability group; however, findings for coordination and variability in both LBP groups were no different from healthy controls (9).

The fear-avoidance model of pain proposes that pain-related fear (10) and pain catastrophizing (11) prompt hypervigilance and avoidance-type behavior, resulting in disability and other negative sequalae (10). This framework is consistent with evidence associating greater pain-related threat (i.e., fear and catastrophizing) and guarded motor behaviors in people with LBP (12). While greater threat may be related to “tight” spinal coordination, this relationship is not studied. Investigating the relationship between pain-related threat and coordination, from the perspective of “tight” and “loose” control, may provide context for divergence of motor behaviors and persistence of symptoms, while helping inform rehabilitation strategies.

Our objective was to estimate the relationship between pain-related threat and spinal coordination amplitude and variability, in adults with chronic LBP, during lifting. A secondary objective was to determine if pain-related threat would differentiate between participants classified as “tight” and “loose” control, in terms of coordination amplitude and variability. We hypothesized that greater pain-related threat would be associated with “tight”, or guarded coordination and variability.

6.3. Methods

6.3.1. Participants

This cross-sectional study recruited participants with chronic non-specific LBP (n = 54) from the community in Montréal, Québec, and the Québec Back Pain Consortium (https://backpainconsortium.ca). Recruitment occurred from October 2020-December 2021.

Participants were screened on the phone for the following criteria: (i) age 18 to 65 years; (ii) pain primarily located between lower ribs and lower buttock, with or without leg pain, lasting for greater than a 3-month period (i.e., chronic non-specific LBP) (13); (iii) daily reports of pain intensity ≥ 2/10 ; (iv) no signs of a serious underlying condition (e.g., cancer), spinal stenosis or radiculopathy, or another specific spinal cause of pain (e.g., fracture) (14); (v) no diagnosis of an inflammatory spinal condition (e.g., ankylosing spondylitis) or widespread pain condition (e.g., fibromyalgia), and (vi) presence of pain-related disability. The latter was screened by asking participants if their pain interfered with their daily life functioning (15). Informed consent was obtained from participants and ethical approval for this study was received from the local research ethics board.

6.3.2. Self-report questionnaires

6.3.2.1. Descriptor variables

Subject demographics, pain duration (date of onset), and number of comorbidities (via pre-determined checklist) were recorded on intake. Pain severity and pain intensity were measured using the Brief Pain Inventory (BPI) (16). Pain during the lifting task was recorded for each repetition using a 0/100 pain intensity scale. Disability was measured using the Oswestry Disability Index (ODI) (17). Physical performance was measured using a 6-minute walk test (18).

6.3.2.2. Pain catastrophizing

Pain catastrophizing refers to an irrational, negative appraisal of pain (11). This construct was measured using the Pain Catastrophizing Scale (PCS) (19). The PCS is a 13-item scale which explores magnification of pain-related threats, feelings of helplessness related to pain, and rumination of pain symptoms. The PCS is a widely used scale in the LBP literature and has good

internal reliability (α = 0.92, 95% confidence interval = 0.91 to 0.93) and test-retest reliability (Spearman ρ = 0.88, 95% confidence interval = 0.83-0.93) (20).

6.3.2.3. Pain-related fear

Pain-related fear refers to fear that emerges when stimuli related to pain are perceived as threatening (10). This construct is measured using an abbreviated 11-item scale (Tampa Scale for Kinesiophobia, TSK-11 (21)), which explores fear-avoidance beliefs relating to both pain and physical activity. This is a widely used scale in the LBP literature with good internal consistency (α = 0.79), test-retest reliability (intraclass correlation coefficient = 0.81), and strong construct validity (21).

6.3.3. Study procedures and lifting task

Study participants were involved in a larger cross-sectional study which included biomechanical data collection during three functional tasks (repeated lifting, repeated bending, blind lifting). When possible, participants completed all functional tasks during testing. Task order was randomized. The present manuscript analyzes the repeated lifting task. All data collection occurred at the Constance Lethbridge-Layton-Mackay rehabilitation centre (Montréal, Québec).

First, a static trial in relaxed standing was recorded, which served as a zero position for spinal joint angles. For the repeated lifting task, the top of the crate (42.0cm x 34.0cm x 29.0cm) was placed at the height of the participant's tibial tubercle and contained weights corresponding to 15% of their body weight. Weights were positioned such that minimal movement occurred. Handles were at the upper part of the crate, on either side. Participants were positioned with their toes touching the lifting platform and arms elevated to 90° of shoulder flexion. Participants were

instructed to lift to waist level while standing fully upright, pause for one second, and replace the crate, without bending their knees (Figure 6.1). No further movement constraints were imposed. Participants were allowed to perform up to three warm-up repetitions to familiarize themselves with the task. Participants were encouraged to complete up to 10 repetitions of the lifting task; however, they could stop at any moment. A researcher (P.I.) observed participant performance of the task and corrected for any deviations from task instructions (e.g., knee bending).

6.3.4. Instrumentation

An electromagnetic motion capture system (TrakSTAR, Ascension Technology, Milton, VT) measured three-dimensional kinematic data of the thigh and spine, sampled at 200 Hz. Sensors were placed at the lateral right thigh, base of the sacrum (S1), and the L3, T12, T9, and C7 spinous processes. Sensors were mounted in custom-molded clips attached to the participants' skin with hypoallergenic double-sided tape. Manufacturer-reported sensor accuracy was 1.4 mm root mean square (RMS) for position and 0.50° RMS for orientation.

6.4. Data processing and analyses

6.4.1. Joint angle calculations and event identification

Kinematic data were smoothed using a low-pass, fourth-order, bidirectional Butterworth filter, with a cut-off frequency of 10 Hz. Orientation data was used to create a [3 x 3] rotation matrix for each segment: right thigh, S1, L3, T12, T9, and C7. To extract inter-segmental joint angles, the rotation matrix of one segment was multiplied by the inverse of the rotation matrix of an adjacent segment (22). The order of rotation was X (sagittal plane), Y (frontal plane), Z (transverse plane). Sagittal plane joint angles were calculated for: (i) the right hip – Hip (S1 relative

to the right thigh); (ii) the lower lumbar spine – LowLx (L3 relative to S1); (iii) and the upper lumbar spine – UpLx (T12 relative to L3). Mean angular velocity was calculated using L3 sensor position data in the anterior-posterior plane for the lifting and replacing periods.

The lifting task was partitioned into lifting and replacing periods. The starting point of the lifting period was defined as velocity of the C7 marker (anterior-posterior direction) first crossing the threshold of $0.1 m/s^2$ for > 1% of signal length. The end point of the lifting period was defined as crossing this threshold, during re-extension with the crate (Figure 6.1). The start of the replacing period was defined as the end of the lifting period and was terminated when the velocity of the C7 marker of crosses the threshold of $0.1 m/s^2$ for > 1% of signal length, for the final time (i.e., during re-extension without the crate). A custom Matlab R2019b script (The MathWorks Inc., Natick, MA, USA) determined all events. All events were visually inspected by plotting the C7 velocity waveform (anterior-posterior direction) alongside sagittal plane hip, lower lumbar, and upper lumbar joint angles, for each individual trial. Events were confirmed by an investigator.

6.4.2. Outcome variables: continuous relative phase analyses

Continuous relative phase analysis described Hip-LowLx and LowLx-UpLx inter-joint coordination amplitude and coordinative variability (23). Mathematical details of our continuous relative phase implementation were described previously (2). In brief, sagittal plane joint angle waveforms during the lifting task, including extraneous data in ranges outside of event identification, were retained. Extraneous data served as data pads, and were used to help manage edge-effects associated with continuous relative phase analyses (24). Phase angles for the LowLx, UpLx, and Hip joints were determined using the Hilbert-transform method (25) and calculated for available trials, for all participants. Next, data pads were removed, leaving one full lifting cycle,

per trial, for analyses. The absolute difference between: (i) Hip and LowLx, and (ii) LowLx and UpLx phase angles determined the continuous relative phase relationship for the Hip-LowLx and LowLx-UpLx joint pairs, respectively. Continuous relative phase values exceeding 180° were subtracted from 360° to manage discontinuities in data (25), restricting values to a scale from 0° (fully in-phase or tightly coupled) to 180° (fully out-of-phase or loosely coupled). All curves were time normalized to 100% of the lifting cycle using a cubic spline and subdivided into lifting and replacing periods based on the above-described events.

The Mean Absolute Relative Phase (MARP) quantified coordination amplitude by determining the ensemble waveform from all available trials for each participant, and then averaging this waveform (26). The Deviation Phase (DP) quantified within-individual coordinative variability by taking the standard deviation of the CRP curve for all available trials for a participant, followed by averaging the standard deviations (26). Calculations of MARP and DP were performed for the Hip-LowLx and LowLx-UpLx joint pairs, in all participants, for both periods (i.e., lifting and replacing).

6.4.3. Statistical analyses

Descriptive statistics for demographic, clinical, and continuous relative phase variables were calculated. Hypothesis-driven, sequential, forward linear regression analyses tested the hypotheses that greater pain catastrophizing and pain-related fear were associated with more in-phase Hip-LowLx and LowLx-UpLx coordination amplitude (MARP) and reduced coordinative variability (DP). Our base model included sex as a covariate, and our independent variables, pain catastrophizing and pain-related fear, were entered as a final step, in separate models. Unstandardized beta coefficients (95% confidence intervals) and p-values were reported. R^2 for

our models were reported alongside r^2 change following the inclusion of pain catastrophizing or pain-related fear. Separate analyses were performed for crate lifting and replacing periods, for both dependent variables (MARP and DP) and joint pairs (Hip-LowLx and LowLx-UpLx), resulting in sixteen regression equations. Statistical significance was set at $P < 0.05$. Assumptions for regression, including multicollinearity, homoscedasticity, residual normality, and linearity, were examined. A causal directed acyclic graph was constructed using free software (DAGitty; http://dagitty.net) and helped identify which covariates should be retained in the statistical model (27). This approach reduces bias by identifying a minimally sufficient adjustment set of covariates for estimating the total effect of an exposure (pain-related threat) on an outcome (coordination amplitude and variability) (27). Potential covariates (sex, age, BMI, current pain intensity, movement speed, negative affect, past injury and pain intensity) were identified based on past literature (28-30) and clinical relevance. Our model and full rationale are included in the Appendix (Appendices 1.9 and 1.10). This process resulted in a statistical adjustment for the sex covariate.

Sample size was determined as described by Milton et al. (31). Past work using similar analyses and variables reported R^2 values ranging from 0.39-0.49 (32), thus we chose an expected R^2 of 0.40. Based on an expected R^2 of 0.40, a desired t-level of 2 (i.e., beta coefficients significant at $P < 0.05$), an r^2 change of 0.05, and 2 variables in our final model, our calculated sample size was 51 participants.

For the lifting period, outliers for Hip-LowLx coordination amplitude (MARP > range+1.5*upper quartile) and variability (DP > range+1.5*upper quartile) were identified. Therefore, for exploratory purposes, we created subgroups based on “loose” (more out-of-phase and more variable) and “tight” (more in-phase and less variable) control phenotypes for LBP. Welch’s t-tests investigated for between-group differences in coordination amplitude (MARP),

variability (DP), pain-related threat, and disability. Statistical significance was set at $P < 0.05$. Analyses were performed in SPSS (v24, IBM Corp., Armonk, USA).

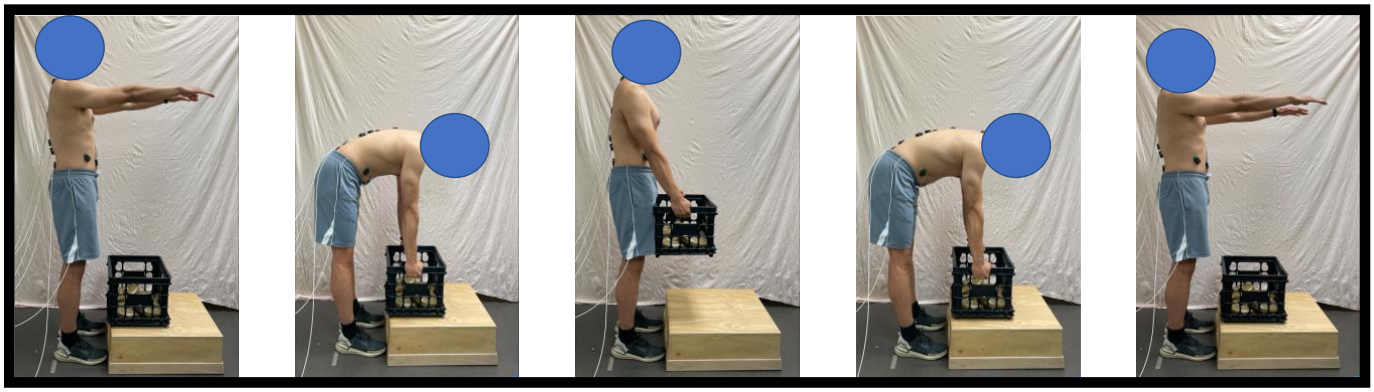

Figure 6.1. Crate lifting task: Crate lifting task corresponding to 15% of participant body weight. From left to right: starting position, bending to lift crate, re-extension with crate, replacing crate, re-extension.

6.5. Results

Overall, 388 potential participants were contacted, 62 were recruited, and 54 participated in data collection. Of these, 5 participants were excluded from analyses based on inability to complete the lifting task (n = 4) and sensor movement during data collection (n = 1). Thus, a total of 49 participants were included in our analyses. Descriptive statistics are listed in Table 6.1 and ensemble averaged curves for coordination are provided in Figure 6.2. "Tight" and "Loose" subgroups are shown in Figure 6.3. Representative participants are shown in appendix 1.9. Statistical assumptions were met, and analyses were deemed appropriate.

6.5.1. Pain catastrophizing and coordination amplitude (MARP) and variability (DP)

For Hip-LowLx coordination amplitude (MARP), the base model (sex) was not significant for both crate lifting ($R^2 = 0.022$, $P = 0.315$) and replacing ($R^2 = 0.026$, $P = 0.265$) periods. Adding pain catastrophizing in a separate step explained unique variance and improved our model for lifting ($R^2 = 0.147$, $P = 0.026$; r^2 change = 0.125, $P = 0.013$) and replacing ($R^2 = 0.136$, $P = 0.034$; r^2 change = 0.099, $P = 0.019$) periods (Table 6.2). These findings underscore a negative relationship, suggesting that greater pain catastrophizing was related to more in-phase coordination (i.e., "tight" joint coupling). For LowLx-UpLx coordination amplitude, our base models (sex) were not statistically significant for lifting ($R^2 = 0.024$, $P = 0.292$) and replacing ($R^2 = 0.017$, $P = 0.372$). Adding pain catastrophizing explained additional variance during lifting (r^2 change = 0.069, $P = 0.068$) and replacing (r^2 change = 0.066, $P = 0.076$); however, these changes and the overall models were not statistically significant (lifting: $R^2 = 0.093$, $P = 0.107$, replacing: $R^2 = 0.083$, $P = 0.137$).

For Hip-LowLx coordinative variability (DP), our base models were not significant for both lifting ($R^2 = 0.073$, $P = 0.060$) and replacing ($R^2 = 0.032$, $P = 0.217$). Adding pain

catastrophizing did not explain a statistically significant amount of variance in variability during lifting (r^2 change = 0.057, P = 0.090, Table 6.2); however, it improved our overall model (R^2 = 0.130, P = 0.041). For crate replacing, pain catastrophizing did not explain additional variance in Hip-LowLx coordinative variability (R^2 change = 0.015, P = 0.402). For LowLx-UpLx coordinative variability, our base model for lifting (R^2 = 0.102, P = 0.026), but not replacing (R^2 = 0.056, P = 0.101), was statistically significant. Adding pain catastrophizing did not explain additional variance during lifting and replacing periods (P > 0.05, Table 6.2).

6.5.2. Pain-related fear and coordination amplitude (MARP) and variability (DP)

For Hip-LowLx coordination amplitude (MARP), adding pain-related fear did not explain unique variance during lifting (r^2 change = 0.072, P = 0.061), or replacing (r^2 change = 0.062, P = 0.083), and the overall models were not statistically significant (lifting: R^2 = 0.094, P = 0.103, replacing: R^2 = 0.089, P = 0.119). For LowLx-UpLx coordination amplitude, pain-related fear did not explain additional variance for lifting and replacing periods (P > 0.05, Table 6.3).

Regarding Hip-LowLx variability (DP), adding pain-related fear did not explain additional variance, in either period. Similarly, for LowLx-UpLx variability, adding pain-related fear did not explain additional variance, in either period (Table 6.3).

6.5.3. Post-hoc analyses

Welsh's t-tests confirmed between-group ("tight", n = 8 and "loose", n = 41) differences in Hip-LowLx coordination amplitude (mean difference = 89.1°) and variability (mean difference = 23.3°), consistent with the proposed phenotypes (P < 0.001), during the lifting period (Table 6.4, Figure 6.3). The "tight" group (i.e., in-phase and less variable) had significantly greater levels of

pain catastrophizing (P = 0.011) and disability (P = 0.013), but not pain-related fear (P = 0.094) or pain intensity (P = 0.086), during lifting (Table 6.4).

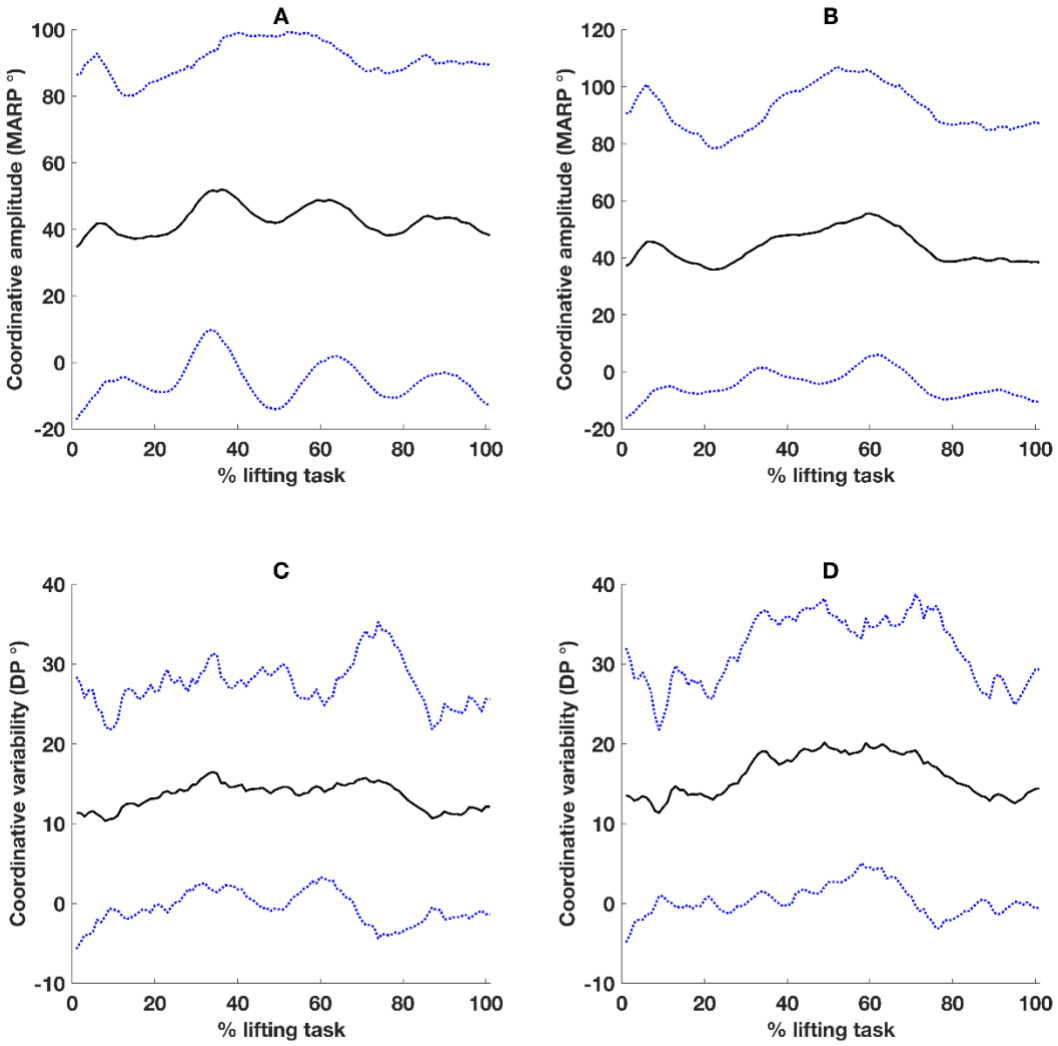

Figure 6.2. Group ensemble averaged continuous relative phase curves for coordination (Mean Absolute Relative Phase, MARP) and variability (Deviation Phase, DP), for the Hip-LowLx and LowLx-UpLx joint pairs, during the full lifting task: A and C represent Hip-LowLx joint pair, while B and D represent LowLx-UpLx joint pair. Solid black lines denote group mean and dotted blue line denotes +/- 1 standard deviation.

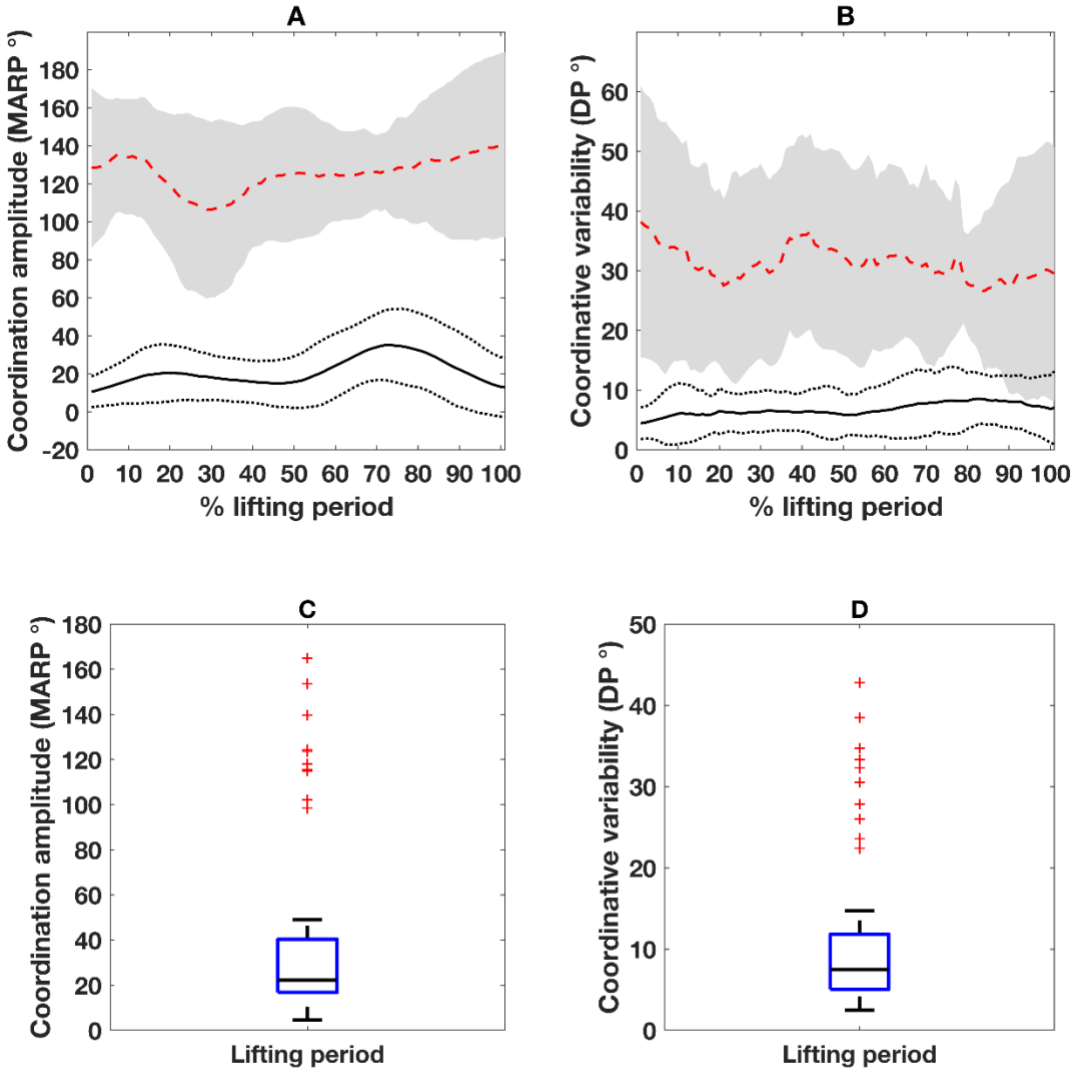

Figure 6.3. Group ensemble averaged continuous relative phase curves and boxplots for Hip-LowLx coordination amplitude (Mean Absolute Relative Phase, MARP) and variability (Deviation Phase, DP) during the lifting period. Greater MARP and DP values reflect more out-of-phase coupling and more variability, respectively. (A-B) Dashed red line corresponds to "loose" subgroup, shaded area represent +/- 1 standard deviation. Solid black line corresponds to "tight" subgroup, dotted lines represent +/- 1 standard deviation. (C-D) Solid black in boxplots lines represent group median and outliers (i.e., "loose" control) are represented by red crosses.

Table 6.1. Descriptive statistics for participant demographics and continuous relative phase variables during a lifting task partitioned into lifting and replacing periods.

Variable	Mean (Standard deviation)
Participants (females)	49 (28)
Age (years)	44.1 (10.8)
BMI	28.9 (7.1)
Pain-related fear (TSK, /44)	28.3 (7.3)
Pain Catastrophizing (PCS, /52)	21.2 (12.4)
Disability (ODI, /100)	21.8 (11.3)
Pain severity (BPI, /10)	3.3 (1.4)
Mean pain during task (/100)	30.4 (18.4)
Hip-LowLx MARP – lift period (°)	42.5 (44.4)
Hip-LowLx MARP – replace period (°)	42.8 (46.8)
Hip-LowLx DP – lift period (°)	11.8 (10.6)
Hip-LowLx DP – replace period (°)	12.2 (12.6)
LowLx-UpLx MARP – lift period (°)	42.4 (44.9)
LowLx-UpLx MARP – replace period (°)	44.3 (46.8)
LowLx-UpLx DP – lift period (°)	14.2 (12.2)
LowLx-UpLx DP – replace period (°)	15.4 (13.5)
Mean velocity (replace, °/s)	22.6 (8.1)
Mean velocity (Lift, °/s)	23.4 (7.3)

BMI: Body Mass Index; TSK: Tampa Scale for Kinesiophobia; PCS: Pain Catastrophizing Scale; ODI: Oswestry Disability Index; BPI: Brief Pain Inventory; MARP: Mean Absolute Relative Phase; DP: Deviation Phase

Table 6.2. Linear regression analyses testing the relationship between pain catastrophizing and Hip-LowLx and LowLx-UpLx coordination amplitude (MARP) and variability (DP) during lifting and replacing periods of a crate lifting task.

Dependent variable	Task period	Variable	β	95% CI	r^2 change (P-value)
Hip-LowLx MARP	Lift	Sex	-9.97	-34.43 to 14.49	
		Pain Catastrophizing	-1.27	-2.25 to -0.29	**0.13 (0.013)**
Hip-LowLx MARP	Replace	Sex	-12.17	-38.11 to 13.77	
		Pain Catastrophizing	-1.26	-2.30 to -0.21	**0.099 (0.019)**
Hip-LowLx DP	Lift	Sex	-5.27	-11.19 to 0.65	
		Pain Catastrophizing	-0.21	-0.44 to 0.033	0.057 (0.090)
Hip-LowLx DP	Replace	Sex	-4.23	-11.55 to 3.10	
		Pain Catastrophizing	-0.12	-0.42 to 0.17	0.015 (0.40)
LowLx-UpLx MARP	Lift	Sex	-11.48	-36.98 to 14.01	
		Pain Catastrophizing	-0.95	-1.98 to 0.072	0.069 (0.068)
LowLx-UpLx MARP	Replace	Sex	-9.87	-36.60 to 16.86	
		Pain Catastrophizing	-0.97	-2.05 to 0.10	0.066 (0.076)
LowLx-UpLx DP	Lift	Sex	-7.70	-14.61 to -0.79	
		Pain Catastrophizing	-0.026	-0.30 to 0.25	0.001 (0.85)
LowLx-UpLx DP	Replace	Sex	-6.54	-14.38 to 1.30	
		Pain Catastrophizing	0.050	-0.27 to 0.37	0.002 (0.75)

Sex is coded as 0 for females and as 1 for males. Unstandardized regression coefficients (β) and 95% confidence intervals from the final model are presented. R^2 change is reported following entering pain catastrophizing in a final step. MARP: Mean Absolute Relative Phase (coordination amplitude). DP: Deviation Phase (coordinative variability). Bolded values denote statistically significant finding ($P < 0.05$)

Table 6.3. Linear regression analyses testing the relationship between pain-related fear and Hip-LowLx and LowLx-UpLx coordination amplitude (MARP) and variability (DP) during lifting and replacing periods of a crate lifting task.

Dependent variable	Task period	Variable	β	95% CI	r^2 change (P-value)
Hip-LowLx MARP	Lift	Sex	-6.50	-32.50 to 19.51	
		Pain-related fear	-1.70	-3.49 to 0.085	0.072 (0.061)
Hip-LowLx MARP	Replace	Sex	-8.81	-36.32 to 18.69	
		Pain-related fear	-1.66	-3.55 to 0.23	0.062 (0.083)
Hip-LowLx DP	Lift	Sex	-5.19	-11.46 to 1.08	
		Pain-related fear	-0.15	-0.58 to 0.28	0.010 (0.49)
Hip-LowLx DP	Replace	Sex	-4.63	-12.25 to 2.98	
		Pain-related fear	0.028	-0.50 to 0.55	<0.001 (0.91)
LowLx-UpLx MARP	Lift	Sex	-10.38	-37.39 to 16.63	
		Pain-related fear	-0.89	-2.74 to 0.97	0.019 (0.34)
LowLx-UpLx MARP	Replace	Sex	-8.28	-36.49 to 19.93	
		Pain-related fear	-1.02	-2.96 to 0.91	0.024 (0.29)
LowLx-UpLx DP	Lift	Sex	-8.39	-15.48 to -1.28	
		Pain-related fear	0.16	-0.33 to 0.65	0.008 (0.51)
LowLx-UpLx DP	Replace	Sex	-7.87	-15.80 to 0.063	
		Pain-related fear	0.38	-0.17 to 0.92	0.038 (0.17)

Sex is coded as 0 for females and as 1 for males. Unstandardized regression coefficients (β) and 95% confidence intervals from the final model are presented. R^2 change is reported following entering pain-related fear in a final step. MARP: Mean Absolute Relative Phase (coordination amplitude). DP: Deviation Phase (coordinative variability). Bolded values denote statistically significant finding ($P < 0.05$)

Table 6.4. Post-Hoc exploratory results for Welch's t-test comparing continuous relative phase and clinical outcomes in "tight" and "loose" subgroups for the Hip-LowLx joint pair in adults with chronic low back pain during the lifting period.

	Loose coordination (n=8)	**Tight coordination (n=41)**	**P-value**	**Mean Difference**
MARP (°)	117.1 (13.0)	28.0 (31.7)	**<0.001**	89.1
DP (°)	31.2 (6.2)	7.9 (6.2)	**<0.001**	23.3
Mean velocity (°/s)	24.0 (7.6)	26.0 (8.3)	0.52	-2.0
BMI	31.6 (10.5)	28.4 (6.3)	0.44	3.2
TSK (/44)	23.8 (7.6)	29.2 (7.0)	0.094	-5.6
PCS (/52)	9.8 (11.4)	23.5 (11.4)	**0.011**	-13.7
Oswestry Disability Index (/100)	14.8 (6.8)	23.2 (11.5)	**0.013**	-8.5
Pain Severity (/10)	2.5 (1.0)	3.4 (1.4)	0.059	-0.9
Mean task pain (/100)	22.3 (12.2)	31.9 (19.1)	0.086	-9.7

Mean Absolute Relative Phase (MARP) reflects coordination amplitude, where greater values suggest out-of-phase joint coupling. Deviation phase (DP) reflects coordinative variability, where greater values indicate greater within-individual variability. Statistical significance was set at $P < 0.05$, significant differences are bolded.

6.6. Discussion

This study investigated the relationship between pain-related threat and Hip-LowLx and LowLx-UpLx coordination amplitude (MARP) and variability (DP), in adults with chronic LBP during lifting. Pain catastrophizing, but not fear, was related to more in-phase ("tight") coordination amplitude for the Hip-LowLx joint pair during lifting and replacing periods. No other statistical relationships were observed. Post-hoc analyses revealed two subgroups broadly consistent with "tight" (more in-phase and less variable) and "loose" (more out-of-phase and more variable) phenotypes (4). These groups were differentiated by levels of pain catastrophizing and disability, with these metrics being greater in the "tight" group. These latter findings remain exploratory and should be interpreted cautiously.

The relationship between greater pain catastrophizing and more in-phase Hip-LowLx coordination amplitude (i.e., "tight" coupling) is consistent with a meta-analysis associating pain-related threat and other measures of guarded motor behavior in people with LBP (e.g., decreased spinal mobility) (33). While we observed a similar trend for an association between pain-related fear and more in-phase coordination, this relationship was not statistically significant. This finding may point to catastrophizing and fear as having distinct relationships with coordination, or that we were underpowered for this comparison (Table 6.3). Pain catastrophizing could relate to more in-phase coordination, in that, greater threat may serve as motivation to stiffen/guard the spine, regardless of the actual effectiveness of this strategy (e.g., sustained "tight" control may lead to compressive loading) (34). Alternatively, elevated catastrophizing may foster a pro-nociceptive nervous system, which could result in the facilitation of pain, thereby prompting a perceived need to guard the spine (35). Few other studies have examined this question from the standpoint of joint coordination, making it difficult to situate our findings. Fujii et al. reported a similar relationship

between greater fear and more in-phase coordination in people with chronic LBP during lifting, albeit in the LowLx-UpLx joint pair (36). While consistent with our findings for the Hip-LowLx joint pair, this diverged from our findings in the LowLx-UpLx joint pair and may be explained by their use of a task-specific measure of fear (0-100 fear scale), differences in task conditions (i.e., no constraint on knee flexion, substantial difference in lifting weight), and/or differences in sample characteristics. Otherwise, our findings were broadly in-line with studies in healthy people undergoing experimentally-induced LBP, whereby greater baseline levels of pain-related threat predicted reduced spinal mobility (37).

We did not find a statistical relationship between threat and coordination variability; however, a trend between greater pain catastrophizing and less variability during crate lifting was observed for the Hip-LowLx joint pair (r^2 change = 0.057, p = 0.090). This may suggest that our analyses were underpowered for this comparison. Again, few comparable studies exist, making situating our work difficult. Veeger et al. reported no relationship between trunk kinematic variability during walking and pain-related fear in people with chronic LBP (38). The lack of a relationship with variability is consistent with our findings, although different procedures were used to quantify variability, and the relative levels of threat during the tasks (walking vs. lifting) may have differentially influenced spinal variability. Otherwise, past work in healthy people showed that negative cognitions predicted reduced variability in postural strategies following experimental back pain (5). However, this was observed from the standpoint of abdominal muscle activity.

This work provides more evidence that threat and motor behaviors consistent with guarding are intertwined (12); however, causation remains unclear. While establishing relationships between variables based on a causal model is a first step in determining causation (Appendix 1.10

and 1.11) (27), our cross-sectional design precludes extracting inferences beyond associations, nor can we exclude the bi-directionality in this relationship (i.e., guarding due to pain leads to greater threat). Further, changes in motor behavior may be influenced by any combination of cortical re-organization, re-enforcement learning, changes in sensory processes, or psychological factors (1, 4, 34); thus, causal mechanisms are unlikely to be unidimensional. Clinically, fleshing out these relationships is important because psychological factors (i.e., pain-related threat) may act as a barrier to successful exercise-based rehabilitation programs, a current staple for managing chronic LBP. From this perspective, integrated care which considers relationship between movement, exercise, and psychological factors may offer better treatment options for LBP, compared to traditional "siloed" approaches (39).

Exploratory post-hoc analyses revealed two presentations in Hip-LowLx coordination consistent with "tight" (in-phase and less variable) and "loose" (out-of-phase and more variable) control, respectively (Figure 6.3). In line with our overarching hypotheses, the "tight" group showed greater pain catastrophizing and disability than the "loose" group, with these changes occurring independent of velocity and pain (Table 6.4). Thus, the "tight" group's negative cognitions appear intertwined with their protective motor strategy, perhaps via learned behaviors (34) or a greater perceived threat of pain (11), and may in-part explain persistence of their motor strategy. In contrast, lower threat and disability in the "loose" group may have encouraged exploration of different movement strategies in which looser coupling, and more variable coordination helped manage spinal load (40). From this standpoint, "loose" control may reflect a more functional movement strategy; however, there is likely a point at which excessive variability becomes problematic and leaves the spine prone to tensile strain due to a transient loss of control (4). While interesting, these inferences remain speculative and further data, including comparisons

with healthy controls, are required to clarify these relationships. Others reported similar findings using experimental LBP in healthy people and observed distinct subgroups of spinal control (stabilized vs. de-stabilized) during a flexion/extension task (41). In-line with our findings, high catastrophizing predicted "tight" control. Future work may consider sampling people with LBP based on these proposed phenotypes; however, to our knowledge, no clinical tests can dissociate between "loose" and "tight" control. Thus, recruitment and study design may prove difficult.

This work has limitations. Skin movement artifacts may have introduced error in lumbo-pelvic kinematics. This resulted in one participant being excluded due to distorted data. Further, the lifting task was difficult for more disabled participants; thus, these findings cannot be generalized to people with high levels of LBP and disability, nor to tasks outside of lifting. Four participants could not complete the task; thus, we did not attain our target sample size of 51 and some analyses may be underpowered. Next, limiting knee flexion during lifting renders the task less natural and can alter movement biomechanics; however, this was done to improve comparability between subjects and foster more threatening task conditions. Fourth, our cross-sectional study design precludes the determination of causation. Lastly, one cannot discount past injury and/or baseline pain severity as a confounder in the relationship between threat and coordination.

6.7. Conclusion

Pain catastrophizing was related to more in-phase Hip-LowLx coordination, but not variability, during a lifting task. This may reflect threat being intertwined with guarded motor behaviors in people with chronic LBP. Preliminary data which sub-grouped participants based on the coordination strategy (in-phase and less variable vs. out-of-phase and more variable) were

similarly differentiated by levels of pain catastrophizing and disability. LowLx-UpLx coordination and variability were unrelated to threat. Future work is required to flesh out causal relationships between variables.

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Chapter 7: General discussion

7.1. Overall summary and integration of findings

7.1.1. Overall summary and integration of findings

The overarching objective of this thesis was to understand changes and divergence in motor behaviors in people with LBP, by studying the determinants of spinal motion and hip-spine coordination. This was investigated from the perspective of (i) biopsychosocial, (ii) task-based, and (iii) methodological factors. The discussion that follows will integrate these findings and consider implications for clinicians and researchers.

Chapter 3 established a small relationship between elevated pain-related threat (via pain-related fear and catastrophizing) and (i) decreased spinal ROM, and (ii) greater trunk muscle activity during functional tasks. This relationship was consistently observed during tasks involving spinal flexion, but not other tasks (e.g., spinal extension). This may reflect pervasive negative connotations surrounding spinal flexion and lifting among the public and health professionals (1). The observed relationship between threat and guarding was somewhat intuitive, as greater pain-related threat may prompt an individual to protect themselves during, or in anticipation of, a painful movement. Causation in this relationship, however, is not established. Moreover, considering pain-related threat's indirect influence on motor behavior (e.g., via possible relationships with learned behaviors, pro-nociceptive mechanisms, and/or cortical re-organization (2, 3)) causation in this relationship is unlikely to be unidimensional.

In chapter 4, we observed differences in coordination in people with LBP at the Hip-LowLx (more variable and more out-of-phase) and the LowLx-UpLx (more out-of-phase) joint pairs, in comparison with healthy controls. This suggested that behaviors consistent with guarding (i.e., more in-phase and less variable) are not observed in all people with LBP, nor do changes in motor behavior occur uniformly across the spine during a sit-to-stand-to-sit task. Partitioning of the task

showed that these changes were accentuated at the start and end of sit-to-stand-to-sit for the Hip-LowLx joint pair, which may reflect greater demands/challenge during specific movement periods. More out-of-phase and variable coordination may underscore poor proprioception in people with LBP. As such, alterations in sensory feedback might result in imprecise motor commands and could expose the spine to excessive tensile strain via uncontrolled movement (3). However, considering the presumedly low threat associated with rising out of a chair and minimal symptom exacerbation during the task, categorizing these behaviors as dysfunctional was not fully warranted. We concluded that future work should investigate coordination and variability during a more threatening task and explore the relationship between coordination and pain-related threat.

Based on findings from chapter 5, where extraneous data have not been collected, spline extrapolation procedures were the most robust approach to control for data distortion associated with continuous relative phase (CRP) analyses. Controlling bias is particularly important for CRP data because these data have qualitative meaning (4). For instance, low CRP values mean more in-phase (i.e., tightly coupled) coordination, while high values are reflective of the opposite behavior. Thus, inflated CRP values could bias one towards the interpretation of more out-of-phase and variable coordination. Our findings informed future work using CRP analyses.

Chapter 6 studied the relationship between pain-related threat (via pain catastrophizing and pain-related fear) and Hip-LowLx and LowLx-UpLx coordination and variability, during a repeated lifting task, in people with chronic LBP. In-line with the results of chapter 3, we associated greater pain catastrophizing (but not fear) with more in-phase Hip-LowLx coordination. While we observed a similar trend with variability, it was not statistically significant. These findings deviated from chapter 4, however, where behaviors consistent with Hip-LowLx "loose" control were observed in our LBP group during sit-to-stand-to-sit. This may relate to differences

in sample characteristics or task demands (e.g., greater threat and challenge during lifting). Post-hoc analyses in chapter 6 revealed two distinct subgroups in our sample that were consistent with proposed phenotypes of "tight" (more in-phase and less variable) and "loose" (more out-of-phase and variable) control for LBP, in the Hip-LowLx joint pair. Subgroups were differentiated by clinical metrics; namely, the "tight" group showed greater levels of pain catastrophizing and disability than the "loose" group. Although these findings were exploratory and should be interpreted cautiously, these data re-enforce the notion of guarded motor behavior and measures of threat being intertwined and may provide context for divergence of findings of coordination and variability in the LBP literature.

7.1.2. Limitations

Specific limitations for each study were described above in corresponding chapters. Limitations generally consistent across all studies will be discussed in the following section. First, biomechanical data were collected using an electromagnetic motion capture system. These systems are prone to electromagnetic interference and skin movement artifacts; thus, these factors may have introduced error in lumbo-pelvic kinematics. To help mitigate this issue, one investigator (P.I.) visually inspected each individual kinematic trial for possible aberrations and removed problematic trials prior to analyses. The two samples of people with chronic LBP analyzed in this thesis showed similar patient characteristics (i.e., low-moderate pain and disability levels) and findings cannot be generalized to people with highly disabling LBP. Further, these findings cannot be generalized beyond the tasks we studied (i.e., sit-to-stand-to-sit and lifting). Considering the task-dependent nature of coordination, interpretation of findings and comparability between studies can be difficult and further research is required to flesh out these relationships. The cross-

sectional nature of our work also precludes any determination of causation. We observed cross-sectional associations in both chapter 4 and chapter 6; however, our data cannot show the direction of these associations. Thus, we need to consider the possibility of the bi-directional nature of these relationships (e.g., painful bending leads to fear of bending). The use of DAGs in chapter 6, however, represent a transparent and important first step in trying to establish causation in this relationship, and support further exploration of this possibility.

7.1.3. Clinical implications

Changes in motor behavior in people with chronic LBP are diverse and may have negative long-term consequences leading to pain and re-injury (3, 5). Chapter 6 observed distinct subgroups of "tight" (in-phase and less variable) and "loose" (out-of-phase and more variable) control, findings consistent with a recent framework proposed by Van Diëen et al. (3). Briefly, "tight" control is proposed to limit movement at the spine at the expense of compressive loading (e.g., guarding/bracing), while "loose" control protects against large muscle forces but may expose the spine to a transient loss of control. In retrospect, this framework extends clinically relevant classification systems for LBP (6, 7), and may better situate results from chapters 3 and 4 within our overarching findings. Therefore, the clinical impact of this work will be discussed using the proposed phenotypes of "loose" and "tight" control in LBP (8).

7.1.3.1. Tight control

Behaviors consistent with "tight" control were observed in chapter 3 (i.e., decreased spinal ROM and greater superficial trunk activity) and chapter 6 (i.e., in-phase and less variable coordination), and these behaviors were associated with indices of greater pain-related threat.

Identifying the presence of this relationship in the clinic, however, may be difficult. First, clinicians could include the use of tools to measure pain-related threat (e.g., Pain Catastrophizing Scale (9)) to help identify the presence of psychosocial distress in people with LBP. Otherwise, task-specific measures of threat (10), or subjective questioning may help flesh out this relationship (11). Although there are no agreed upon clinical tests to determine "tight" control, rehabilitation professionals could be watchful for reduced spinal ROM (12), increased superficial trunk muscle activity (e.g., bracing and breath holding) (13), and/or slow and tightly coordinated (i.e., overly stiff) movement (3). These behaviors are thought to be consistent with a phenotype of "tight" control; however, a framework for reliably identifying this behavior in clinic requires further study. Our findings suggest that "tight" control tends to occur during flexion-based tasks and may be accentuated during challenging aspects of functional movement. Thus, clinicians are encouraged to "break down" a problematic task into different components (e.g., lifting a box vs. replacing a box) and analyze these movement periods separately. Lastly, we observed that dysfunction is more likely to occur at the Hip-LowLx joint pair; therefore, clinical assessment could be focused on this region. Taken together, these concepts may assist clinicians in identifying when "tight" control and psychological distress might need to be considered together.

"Tight" control is in-line with existing treatment paradigms for LBP; namely the hypomobility category from the Treatment-Based Classification system (6), or the movement impairment category from the O'Sullivan classification system (7). From this perspective, changing motor behavior associated with "tight" control may involve any combination of manual/manipulative spinal therapy, superficial trunk relaxation, and lumbo-pelvic dissociation (e.g., hip-hinging) (6, 7). Changing coordination and increasing variability, could involve coaching the patient to lift, bend, or function from a variety of postures and positions while moving away of

the notion of “optimal” posture or lifting technique (14). However, targeting movement-related impairments is not sufficient. Clinicians should also be adept in reducing threat-value of pain, either via behavioral experiments (15) or pain education (16). Integrated treatment frameworks which consider movement dysfunction and psychological impairments in tandem, such as cognitive functional therapy, may help manage “tight” control and associated psychological distress from a biopsychosocial perspective (15).

7.1.3.2. Loose control

Chapter 4 (and to a lesser extent chapter 6) revealed a subgroup of people with LBP showing “loose” control (i.e., more out-of-phase and variable coordination). Preliminary findings suggest that this group may be characterized by lower levels of pain-related threat than the “tight” control phenotype. While this is intuitive (i.e., low threat is associated with less guarded movement), this notion requires further study. “Loose” control is aligned with the “stabilization” category for the Treatment Based Classification system and “motor control impairment” of the O’Sullivan Classification System (6, 7). Broadly, “loose” control is characterized by aberrant spinal motion, excessive spinal segmental rotation, proprioceptive deficits, reduced deep abdominal activity, and is thought to reflect less robust control of the spine (8). Thus, this group may benefit from targeted muscle activation, while working towards functional control of a neutral spine during movement (6, 7). Clinical tests may help identify this subgroup (17); however, reliably identifying “loose” control remains challenging. Like “tight” control, clinicians could focus assessment on the Hip-LowLx joint pair and during challenging aspects of movement. Additionally, we must consider that “tight” and “loose” control occur on a spectrum of motor behaviors which includes a subgroup of people with LBP who have “normal” control (3, 18). This

"normal" group may be characterized by non-nociceptive pain mechanisms (i.e., movement dysfunction is less relevant) and might be better managed using psychologically-informed physiotherapy (19). While classifying people with LBP based on their proposed control phenotype may help direct care, these suggested treatments are speculative and require formal testing. Further, despite "loose" and "tight" control phenotypes being evidence-informed, they remain theoretical and are lacking validation. Lastly, the lack of reliable diagnostic criteria and clear "cut off" points between normal and dysfunctional control remains a gap that requires addressing.

7.1.4. Implications for researchers and future direction

Chapter 4 showed that the lumbar spine behaves as two distinct regions, and that this behavior changes dependent on movement period. Future work could consider incorporating these notions which may increase sensitivity in identifying between-group differences in neuromuscular impairments in people with LBP. Further, the choice of data processing techniques common to continuous relative phase analyses can impact subsequent findings. While current guidelines suggest the methods used herein (i.e., Hilbert-transform approach (20)), chapter 5 extended these practices by proposing a solution to control data distortion associated with this approach. Kinematic data collection is often "messy", and adoption of best practices will help with interpretability of data.

Chapter 6 showed distinct sub-groups in our sample that were differentiated by Hip-LowLx coordination during lifting. While these findings were exploratory, researchers could consider data from both a group and individual level, a practice which may help reconcile heterogeneity in LBP. Future work could attempt to differentiate between these proposed subgroups based on clinical tests or validate the existence of these phenotypes.

We have shown an association between pain-related threat and various measures of movement biomechanics (chapter 3 and 6). Thus, researchers are encouraged to consider the possible influence of threat on movement biomechanics and incorporate appropriate measures (e.g., Pain Catastrophizing Scale) into study design. Future work could study causal relationships between pain-related threat and guarded motor behavior. This could involve sampling people with LBP based on their motor behavior (i.e., "tight" vs. "loose" coordination) and performing group comparisons across a battery of functional tasks; however, clinical tests to distinguish the two groups are lacking.

Lastly, investigating the impact of clinically relevant biopsychosocial variables (e.g., pain sensitivity) on coordination or using novel methods (e.g., statistical parametric mapping) may help us understand how motor behavior is changed in people with LBP.

7.2. Chapter 7 references

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Chapter 8: Conclusion and summary

8.1. Conclusion and summary

This dissertation aimed to understand changes and divergence in motor behaviors in people with LBP, by studying biopsychosocial, task-based, and methodological determinants of spinal motion and coordination. First, a meta-analysis associated greater pain-related threat (pain catastrophizing and pain-related fear) and guarded motor behavior (decreased spinal ROM and increased trunk muscle activity) in people with LBP, particularly during flexion tasks. Next, more out-of-phase coordination amplitude and variability were observed at the Hip-LowLx joint pair during challenging aspects of a sit-to-stand-to-sit task, compared to healthy controls. A methodological study established a spline extrapolation data padding procedure to control for data distortion associated with continuous relative phase (i.e., coordination) analyses. Last, greater pain catastrophizing, but not fear, was associated with more in-phase Hip-LowLx coordination during a lifting task in people with chronic LBP. Exploratory analyses revealed distinct subgroups (i.e., "tight" and "loose" control) which were differentiated by pain catastrophizing and disability.

Altogether, this work has shown that when investigating spinal mobility and coordination, distinct presentations in motor behavior may occur across similar samples. Therefore, to understand and draw meaningful clinical inferences from these observations, findings should be considered in the context of the possible impact of pain-related threat, methodological procedures selected to carry out the investigation, and task selection and conditions. Considered together, this may have implications for the management of LBP; however, additional research is needed to guide clinical care.

Appendix

Appendix 1.1. Medline Search Strategy

1. Low Back Pain/
2. Back Pain/
3. 1 or 2
4. Biomechanical Phenomena/
5. Abdominal Muscles/
6. Electromyography
7. Muscle, Skeletal/
8. Muscle Contraction/
9. Movement/
10. 4 or 5 or 6 or 7 or 8 or 9
11. Fear/
12. Catastrophizing/
13. Anticipation, Psychological/
14. Phobic Disorders/
15. 11 or 12 or 13 or 14
16. (low back pain or back pain or non specific low back pain or lumbago or chronic low back pain or dorsalgia or lumbar pain or lumbar strain or lumbar sprain or disc herniation or stenosis or sciatica or spondylolysis or spondylolisthesis).tw,kf.
17. (biomechanical phenomena or abdominal muscles or electromyography or skeletal muscle or muscle contraction or movement or emg or bracing or guarding or muscle activity or lumbar motion or lumbar kinematics or lumbar mobility or trunk stability or biomechanics or spine loading or lumbar kinetics or coordination or variability).tw,kf.
18. (fear or catastrophizing or anticipation or phobic disorders or fear avoidance or fear avoidance beliefs of kinesiophobia or pain related fear or pain catastrophizing or psychological factors or psychosocial factors).tw,kf.
19. (3 or 16) and (10 or 17) and (15 or 18)

Appendix 1.2. Summary of risk of bias scoring using Newcastle-Ottawa Scale for cross-sectional studies

	Selection				Comparability		Outcome		Total (/10)	Summary
Study	**Represent-ativeness of the sample**	**Sample size**	**Non-respondents**	**Ascertainment of exposure**	**Control for pain**	**Controlled for other factor**	**Assessment of outcome**	**Statistical test**		
Alschuler (2009)	C (0)	C (0)	C (0)	A (2)	X (0)	B (1)	B (2)	B (0)	6	S
Alcaraz-Clariana (2020)	B (1)	A (1)	A (1)	A (2)	X (0)	B (1)	B (2)	B (0)	8	G
Alsubaie (2021)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Demoulin (2013)	B (1)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	B (0)	7	G
Dubois (2014)	B (1)	C (0)	C (0)	B (1)	A (1)	B (1)	B (2)	B (0)	6	S
Geisser (2004)	C (0)	C (0)	C (0)	A (2)	X (0)	B (1)	B (2)	B (0)	6	S
George (2006)	B (1)	C (0)	C (0)	A (2)	X (0)	X (0)	B (2)	A (1)	6	S
Grotle (2004a, 2004b)	C (0)	C (0)	A (1)	A (2)	X (0)	X (0)	B (2)	B (0)	5	S
Jette (2016)	C (0)	A (1)	A (1)	A (2)	X (0)	X (0)	B (2)	A (1)	7	G
Kernan (2007)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	B (0)	6	S
Kim (2017)	B (1)	A (1)	A (1)	A (2)	X (0)	B (1)	B (2)	B (0)	8	G

LaTouche (2019)	C (0)	C (0)	A (1)	A (2)	X (0)	X (0)	B (2)	B (0)	5	S
Lamoth (2006)	B (1)	B (0)	C (0)	A (2)	X (0)	B (1)	B (2)	B (0)	6	S
Lima (2018)	B (1)	A (1)	A (1)	A (2)	X (0)	X (0)	B (2)	A (1)	8	G
Mannion (2001)	B (1)	C (0)	B (0)	B (1)	X (0)	B (1)	B (2)	B (0)	5	S
Marich (2017)	C (0)	A (1)	A (1)	A (2)	A (1)	B (1)	B (2)	B (0)	8	G
Masse-Alarie (2016)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Matheve (2019)	A (1)	C (0)	A (1)	A (2)	A (1)	B (1)	B (2)	B (0)	8	G
McCraken (1998)	C (0)	C (0)	C (0)	A (2)	A (1)	X (0)	D (0)	B (0)	3	US
Nieto-Garcia (2019)	C (0)	C (0)	A (1)	A (2)	X (0)	X (0)	B (2)	B (0)	5	S
Osumi (2019)	B (1)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	8	G
Pagé (2015)	B (1)	A (1)	A (1)	A (2)	X (0)	X (0)	B (2)	B (0)	7	G
Pakzad (2016)	B (1)	C (0)	A (1)	A (2)	A (1)	B (1)	B (2)	B (0)	8	G
Salt (2020)	B (1)	C (0)	B (0)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Svendsen (2013)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Thomas (2008)	C (0)	C (0)	B (0)	A (2)	X (0)	B (1)	B (2)	B (0)	5	S

Thomas (2007)	B (1)	C (0)	B (0)	A (2)	X (0)	X (0)	B (2)	B (0)	5	S
Vaisy (2015)	B (1)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	B (0)	7	G
Van der Hulst (2010a)	C (0)	B (0)	A (1)	B (1)	A (1)	B (1)	B (2)	B (0)	6	S
Van der Hulst (2010b)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Veeger (2020)	C (0)	A (1)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	8	G
Vincent (2011)	C (0)	C (0)	A (1)	A (2)	X (0)	B (1)	B (2)	A (1)	7	G
Watson (1997)	C (0)	C (0)	A (1)	A (2)	X (0)	X (0)	B (2)	A (1)	6	S

VG: Very good 9-10 points (n=0); G: Good 7-8 points (n=18); S: Satisfactory 5-6 points (14); US: Unsatisfactory 0-4 points (n=1)

Appendix 1.3. Newcastle Ottawa Scale and scoring criteria

The Newcastle-Ottawa scale utilizes a scoring system which evaluates studies on selection and comparability of the groups, and ascertainment of the exposure and outcome of interest. A maximum score of 10/10 is possible. Studies were scored out of ten and categorized as very good (9-10 points), good (7-8 points), satisfactory (5-6 points) and unsatisfactory (0-4 points). Our interpretation and scoring instructions are listed in blue.

Selection: (Maximum 5 stars)

1. Representativeness of the sample:
    a. Truly representative of the average in the target population. * (all subjects or random sampling)
    b. Somewhat representative of the average in the target group. * (non-random sampling)
    c. Selected group of users/convenience sample.
    d. No description of the derivation of the included subjects.

Our studies are not dealing with all subjects of a target population and rarely employ random sampling. Thus, there will not be many studies scored as "a". Studies recruited from a combination of the community, university centres and clinics etc… will be listed as "b". Studies utilizing a convenience sample from one site or lacking proper description will be scored as "c" and "d", respectively.

2. Sample size:
    a. Justified and satisfactory (including sample size calculation). *
    b. Not justified.
    c. No information provided

Only studies to report a justified sample size (and calculations) will be scored as "a". Studies not properly justified will be scored "b", while "c" will be allocated if no information is provided.

3. Non-respondents:
    a. Proportion of target sample recruited attains pre-specified target or basic summary of non-respondent characteristics in sampling frame recorded. *
    b. Unsatisfactory recruitment rate, no summary data on non-respondents.
    c. No information provided

This is difficult to assess in cross-sectional studies. If studies said they recruited X and there are X in demographic table, we gave it to them. Non-response bias occurs when people who refuse to take part in a study, or those who drop out before the study is completed, are systematically different from those who participate.

4. Ascertainment of the exposure (risk factor):
    a. Validated measurement tool. **

b. Non-validated measurement tool, but the tool is available or described. *
c. No description of the measurement tool.

Accepted, valid tools for pain-related fear: TSK, FABQ, PHODA, PHODA-Sev, PASS
Accepted, valid tools for pain catastrophizing: PCS, CSQ-cat.
'b' was scored when authors used a principal component analysis to summarize multiple psychological factors

Comparability: (Maximum 2 stars)

1. The subjects in different outcome groups are comparable on the basis of design, or analyses (i.e., confounding factors in the relationship between pain-related threat and motor behavior are controlled for.)
    a. The study controls for the most important factor (Pain). *
    b. The study controlled for any additional factor (e.g., age, sex, movement speed, disability). *

One star allocated for controlling for most important factor – pain. Another star for controlling for demographics (age, sex), or other relevant factors. Simply standardizing the task is not enough.

Outcome:

1. Assessment of outcome:
    a. Assessment using objective validated laboratory methods. **
    b. Unblinded assessment using objective validated laboratory methods. **
    c. Used non-standard or non-validated laboratory methods. *
    d. No description/non-standard laboratory methods used.

Self-explanatory.

2. Statistical test:
    a. Statistical test used to analyse the data clearly described, appropriate and measures of association presented including confidence intervals and probability level (p value). *
    b. Statistical test not appropriate, not described or incomplete.

Given most outcomes were correlation scores, confidence intervals are quite rare. We judged on reporting of full p-value. If exact p-value was not stated (e.g. p>0.05) we scored them a "b".

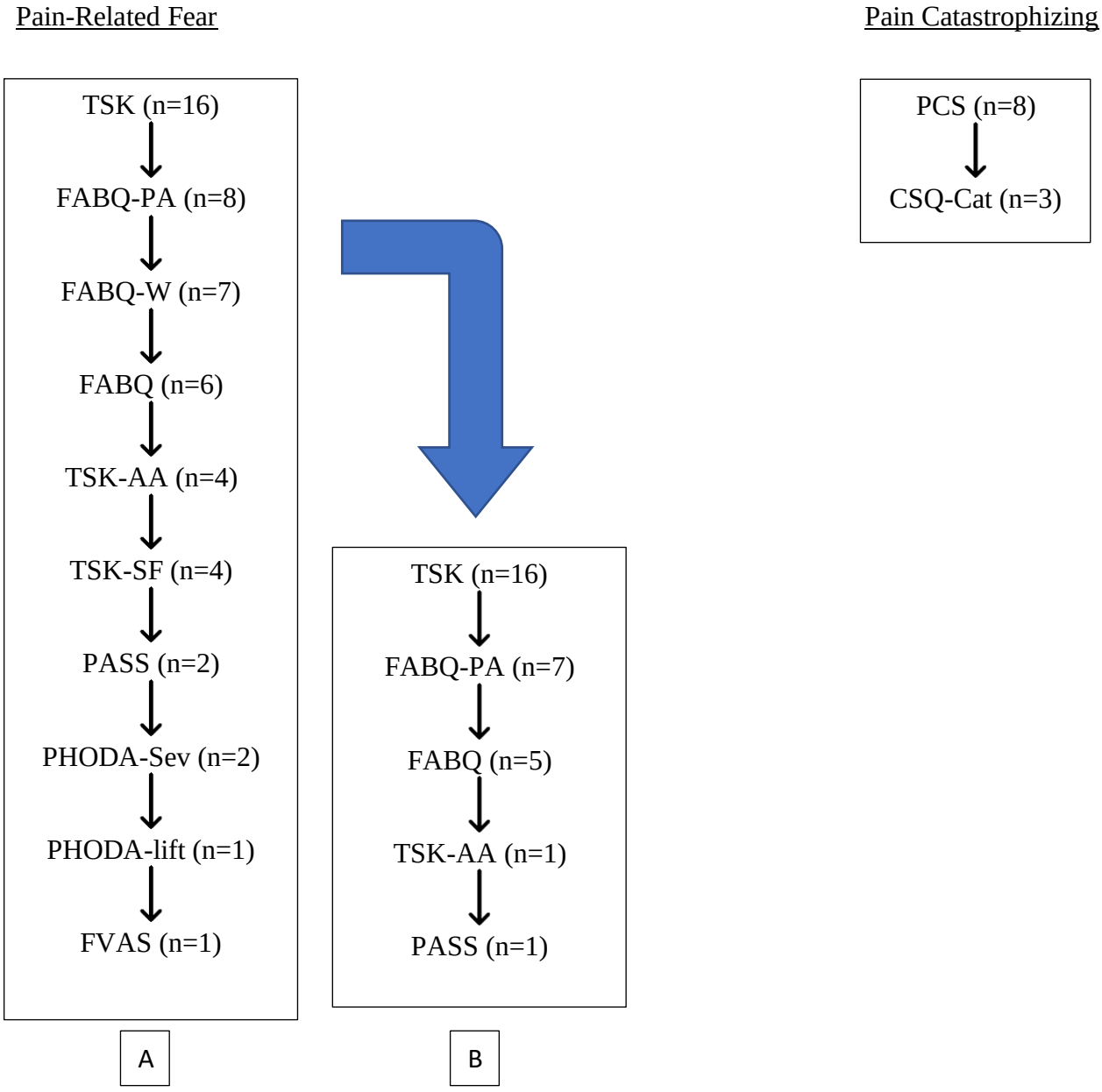
Final Scoring: Very Good Studies: 9-10 points ; Good Studies: 7-8 points; Satisfactory Studies: 5-6 points; Unsatisfactory Studies: 0 to 4 points
This scale has been adapted from the Newcastle-Ottawa Quality Assessment Scale for cohort studies to provide quality assessment of cross-sectional studies[1].

[1] Herzog R, et al. Is Healthcare Workers' Intention to Vaccinate Related to their Knowledge, Beliefs and Attitudes? A Systematic Review. *BMC Public Health* 2013 **13**:15

Appendix 1.4. Percent agreement for Newcastle-Ottawa Scale scoring between raters

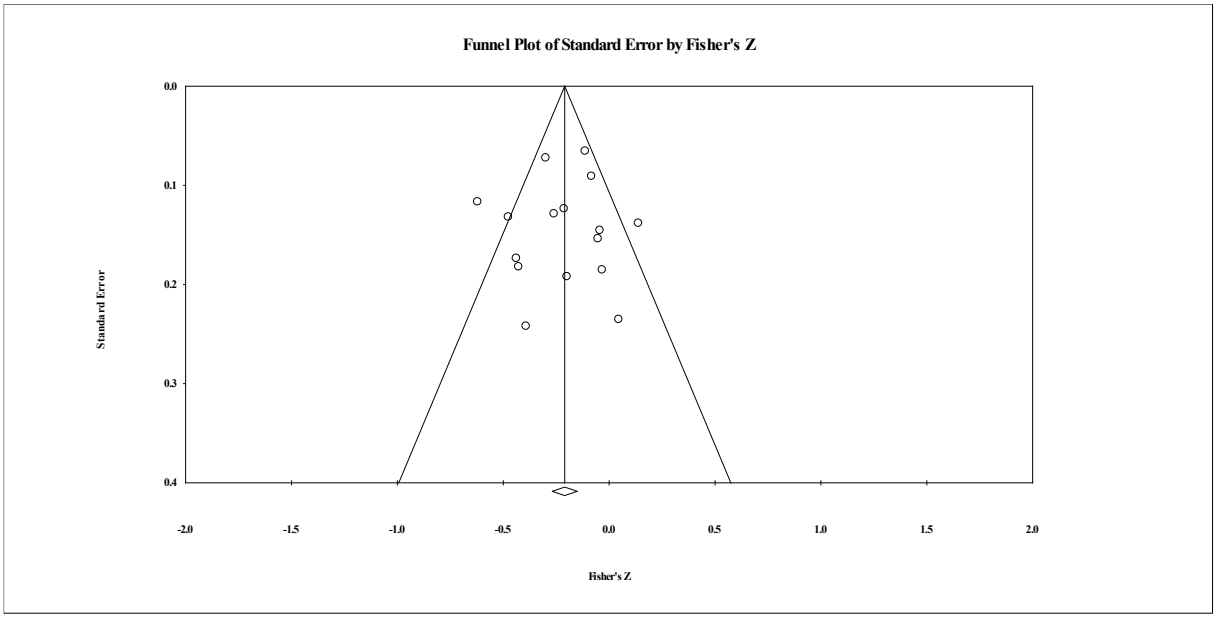
		Disagreements	% Agreement
Selection 1	Representativeness of the sample:	3/34	91.2
Selection 2	Sample Size	3/34	91.2
Selection 3	Non-respondents	0/34	100
Selection 4	Ascertainment of exposure	0/34	100
Comparability 1a	Control for confounder 1 (pain)	7/34	79.4
Comparability 1b	Control for confounder 2 (various)	6/34	82.4
Outcome 1	Ascertainment of outcome	0/34	100
Outcome 2	Statistical test	4/34	88.2

Appendix 1.5. Decision making algorithm for selecting primary psychological measure used in analyses

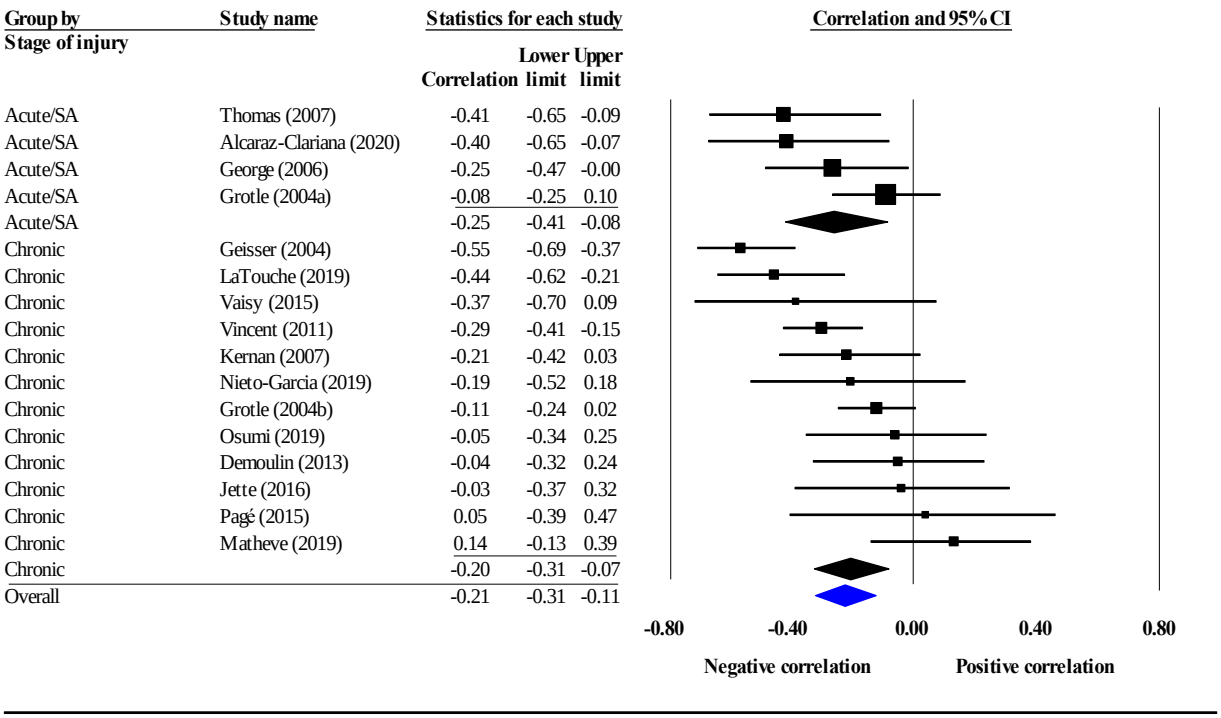


Appendix B. Box (A) indicates the number of times each tool appeared across our studies, including those which reported multiple measures for fear. Box (B) represents the results of our decision-making process based on frequency of use. There was no overlap for pain catastrophizing, thus, this process was not required.

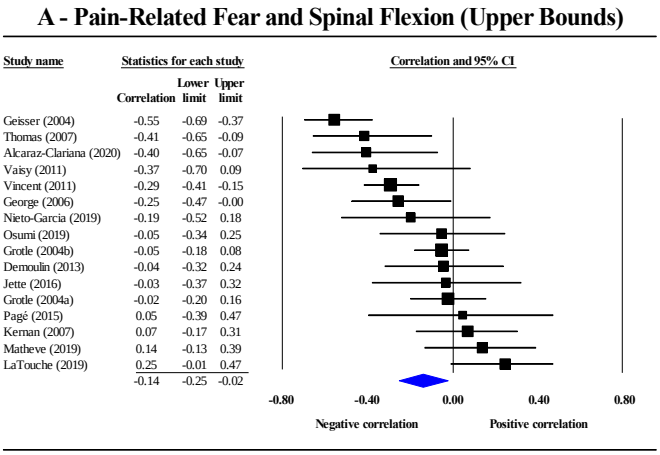
TSK: Tampa Scale for Kinesiophobia; TSK-AA: Tampa Scale for Kinesiophobia Activity Avoidance subscale; TSK-SF: Tampa Scale for Kinesiophobia Somatic Focus subscale FABQ: Fear-Avoidance Beliefs Questionnaire; FABQ-PA: Fear-Avoidance Beliefs Questionnaire Physical-Activity subscale; FABQ-W: Fear-Avoidance Beliefs Questionnaire Work subscale; PASS: Pain Anxiety Symptoms Scale; PHODA-Sev: Photograph Series of Daily Activities Short Electronic version; PHODA-lift: Photograph Series of Daily Activities Lifting image; FVAS: Fear Visual Analog Scale; PCS: Pain Catastrophizing Scale; CSQ-Cat: Coping Strategies Questionnaire Catastrophizing subscale.

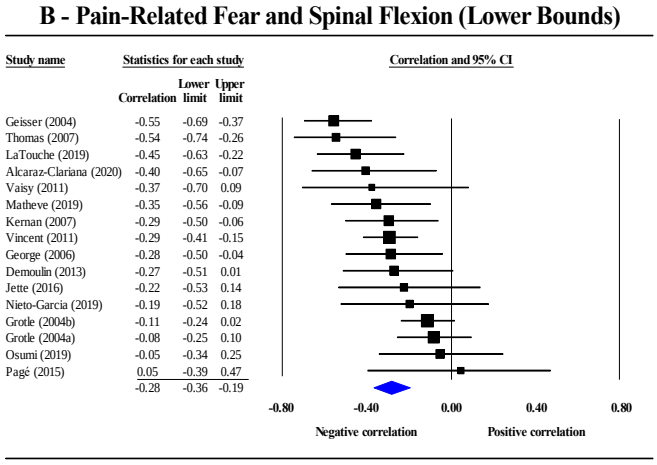


Appendix 1.6. Funnel plot for publication bias: Funnel plot showing standard error by Fisher's Z for the relationship between pain-related fear and spinal flexion range of motion (n=16).



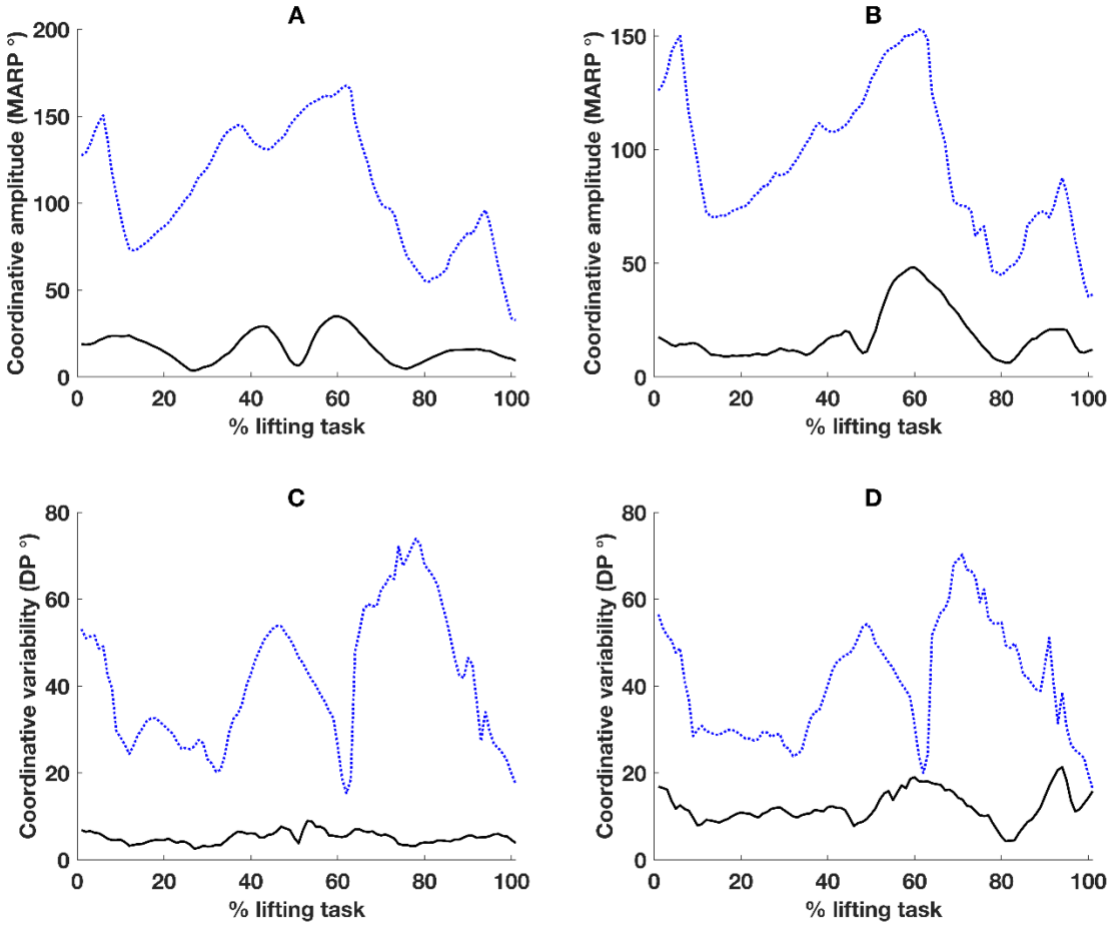
Appendix 1.7. Forest plot (subgroup analyses) for pain-related fear and spinal flexion: Subgroup analyses (acute/sub-acute vs. chronic) examining the relationship between spinal flexion and pain-related fear during flexion tasks. Acute/sub-acute and chronic subgroups demonstrate a small negative association, respectively.

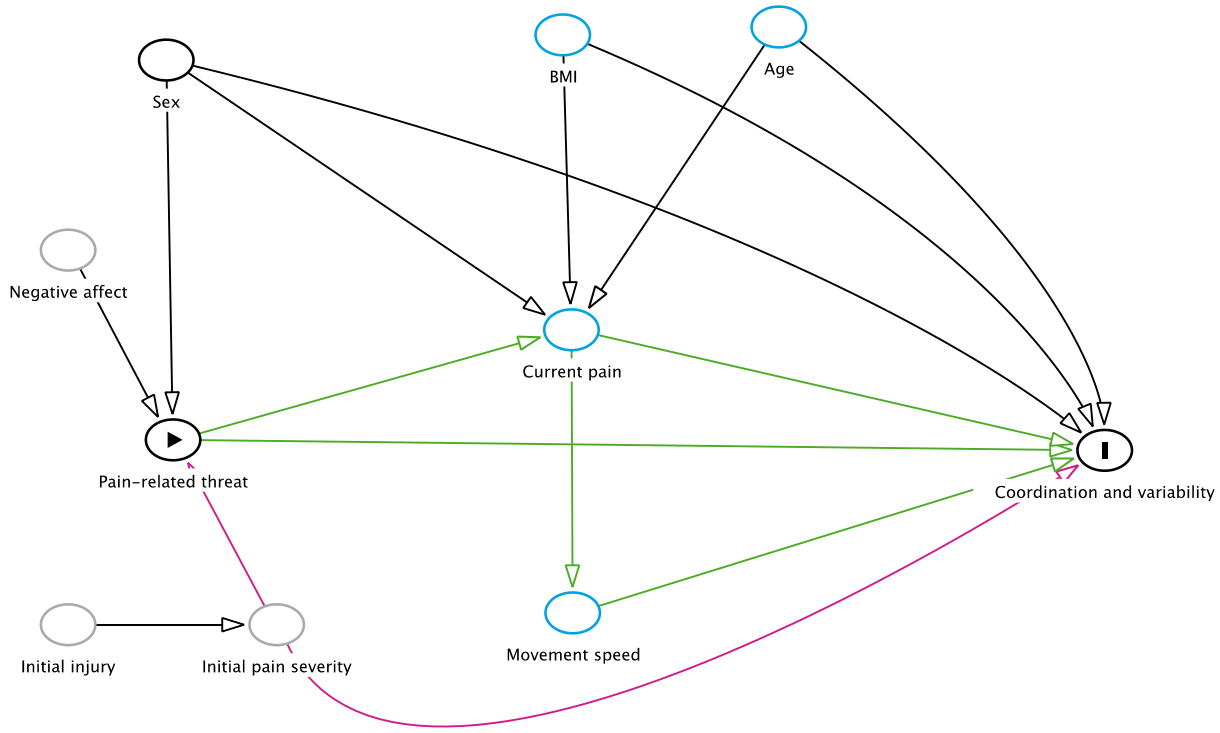




Appendix 1.8. Forest plot (sensitivity analyses) for pain-related fear and spinal flexion: A: Upper bound sensitivity analyses (correlations showing strongest association in the positive direction) of the relationship between pain-related fear and spinal flexion range of motion during flexion tasks, in acute/sub-acute and chronic low back pain. Synthesis shows no correlation. B: Lower bound sensitivity analyses (correlations showing strongest association in the negative direction) of the relationship between pain-related fear and spinal flexion range of motion during flexion tasks, in acute/sub-acute and chronic low back pain. Synthesis shows a moderate negative correlation.

Appendix 1.9. Mean continuous relative phase curves for coordination (Mean Absolute Relative Phase, MARP) and variability (Deviation Phase, DP), for the Hip-LowLx and LowLx-UpLx joint pairs, during the full lifting task for representative participants corresponding to "tight" and "loose" subgroups: A and C represent Hip-LowLx joint pair, while B and D represent LowLx-UpLx joint pair. Solid black lines denote a "tight" participant and dotted blue lines denote a "loose" participant.





Appendix 1.10. Causal directed acyclic graph: Causal directed acyclic graph (DAG) used to estimate the total effect of pain-related threat (exposure) on coordination and variability (outcome) while adjusting for the minimally sufficient set of covariates (sex). Green paths indicate causal effect, while purple paths indicate confounding. Black paths are not on the hypothesized causal pathway. Grey variables are latent (unmeasured variables). White variables indicate a covariate that has been controlled for statistically (sex). Blue variables are ancestors of our outcome.

Appendix 1.11. Model rationale for directed acyclic graph.

Creating a causal Directed Acyclic Graphs (DAGs) is a transparent and robust approach used to identify adjustment sets of covariates for estimating the effect of an exposure (pain-related threat) on a dependent variable (spinal coordination and variability). Traditional methods of covariate selection may increase bias rather than controlling for it; thus, the use of DAGs is a recommended approach for covariate selection in causal models (1).

- Potential covariates of sex, age, BMI, movement speed, and current pain were included in the model based on past literature showing their association with spinal mobility and/or joint coordination (2-6).
- Considering evidence that women experience more LBP than men; LBP is more common in women and in older adults; and the strong links between LBP and obesity; paths were drawn showing the association between these variables and current pain intensity (7-9).
- It appears there is some evidence of sex differences on levels of pain catastrophizing; thus, a path was drawn connecting sex and pain-related threat (10).
- Initial injury and pain severity were included as latent (unmeasured) variables in the model due to their potential confounding effects. For instance, an acute injury could cause severe pain initially, which may independently influence both pain-related threat (i.e., intense pain leads to greater threat of pain) and motor behavior (i.e., intense pain leads to guarded motor behavior).
- Negative affective states (e.g., depression and anxiety) was included as a latent variable due to pain catastrophizing sharing significant variance with broader negative affect constructs (11).

- A path was drawn connecting pain and movement speed based on evidence that, on average, people with LBP move slower than healthy controls (12).

Final model: Based on the DAG approach, the minimal sufficient adjustment set for estimating the total effect of pain-related threat on coordination and variability was the sex covariate.

Appendix 1.12. Ethics certificate

Comité d'éthique de la recherche
des établissements du CRIR

CRIR

Certificat d'éthique

Par la présente, le comité d'éthique de la recherche des établissements du CRIR (CÉR) atteste qu'il a évalué, lors de sa réunion du 9 juillet 2019, le projet de recherche **CRIR-1414-0319** intitulé :

« Why do people with chronic low back pain move differently? The influence of biopsychosocial variables on movement variability».

Présenté par: **Shawn Robbins, Ph.D**
Patrick Ippersiel, candidat au

Le présent projet répond aux exigences éthiques de notre CÉR. Le Comité autorise donc sa mise en œuvre sur la foi des documents suivants :

- **Lettre de présentation au CÉR datée du 7 mai 2019;**
- **Formulaire A;**
- **Formulaire d'évaluation du Centre de réadaptation Lethbridge-Layton-Mackay du CIUSSS Centre-Ouest-de-l'Île-de-Montréal daté du 7 juin 2019, mentionnant que le projet est acceptable sur le plan de la convenance institutionnelle;**
- **Évaluation scientifique du projet de recherche du 7 mai 2019 effectuée par le Comité d'évaluation scientifique du CRIR;**
- **Correspondance du 5 juin 2019 du président du Comité d'évaluation scientifique du CRIR reconnaissant l'évaluation scientifique du projet de recherche effectuée par le comité d'évaluation scientifique de l'Université McGill;**
- **Budget;**
- **Protocole de recherche (version du 12 août 2019);**
- **Formulaires d'information et de consentement français et anglais (version du 12 août 2019);**
- **Formulaires démographiques français et anglais;**
- **Affiches de recrutement (versions française et anglaise du 12 août 2019);**
- **Questionnaires de santé d'éligibilité (versions française et anglaise du 12 août 2019);**
- **Questionnaires utilisés :**
  - **Tampa Scale of Kinesiophobia (versions française et anglaise du 12 août 2019);**
  - **Pain Catastrophizing Scale (versions française et anglaise du 12 août 2019);**
  - **Oswestry Disability Index (versions française et anglaise du 12 août 2019);**
  - **Start Back Screening Tool (versions française et anglaise);**
  - **Brief Pain Inventory (versions française et anglaise du 12 août 2019);**
  - **Patient Health Questionnaire (versions française et anglaise du 12 août 2019);**
  - **Central Sensitization Inventory (versions anglaise seulement ; version française à venir et demandée par le chercheur aux auteurs du questionnaire).**

Ce projet se déroulera dans le site du CRIR suivant :

- **Centre de réadaptation Lethbridge-Layton-Mackay du CIUSSS du Centre-Ouest-de-l'Île-de-Montréal (installation Constance-Lethbridge)**

Ce certificat est valable pour un an. En acceptant le présent certificat d'éthique, le chercheur s'engage à :

1. Informer, dès que possible, le CÉR de tout changement qui pourrait être apporté à la présente recherche ou aux documents qui en découlent (Formulaire M) ;
2. Notifier, dès que possible, le CÉR de tout incident ou accident lié à la procédure du projet ;
3. Notifier, dès que possible, le CÉR de tout nouveau renseignement susceptible d'affecter l'intégrité ou l'éthicité du projet de recherche, ou encore, d'influer sur la décision d'un sujet de recherche quant à sa participation au projet ;
4. Notifier, dès que possible, le CÉR de toute suspension ou annulation d'autorisation relative au projet qu'aura formulée un organisme de subvention ou de réglementation ;
5. Notifier, dès que possible, le CÉR de tout problème constaté par un tiers au cours d'une activité de surveillance ou de vérification, interne ou externe, qui est susceptible de remettre en question l'intégrité ou l'éthicité du projet ainsi que la décision du CÉR ;
6. Notifier, dès que possible, le CÉR de l'interruption prématurée, temporaire ou définitive du projet. Cette modification doit être accompagnée d'un rapport faisant état des motifs à la base de cette interruption et des répercussions sur celles-ci sur les sujets de recherche ;
7. Fournir annuellement au CÉR un rapport d'étape l'informant de l'avancement des travaux de recherche (formulaire R) ;
8. Demander le renouvellement annuel de son certificat d'éthique ;
9. Tenir et conserver, selon la procédure prévue dans la *Politique portant sur la conservation d'une liste des sujets de recherche*, incluse dans le cadre réglementaire des établissements du CRIR, une liste des personnes qui ont accepté de prendre part à la présente étude ;
10. Envoyer au CÉR une copie de son rapport de fin de projet / publication ;
11. En vertu de l'article 19.2 de la *Loi sur les services de santé et les services sociaux*, obtenir l'autorisation du Directeur des services professionnels de l'établissement sollicité avant d'aller consulter les dossiers des usagers de cet établissement, le cas échéant.

Me Michel T. Giroux
Président du CÉR

Date d'émission
12 août 2019

Centre intégré universitaire de santé et de services sociaux du Centre-Ouest-de-l'Île-de-Montréal
Québec
Integrated Health and Social Services University Network for West-Central Montreal

2019-09-19

Mr Shawn Robbins
c/o: Mushirah Hossenbaccus
email: m.hossenbaccus.clethb@ssss.gouv.qc.ca

Object: Project MEO-50-2020-1979 - Authorization to conduct your research study at WCMH.
Why do people with chronic lower back pain move differently? The influence of biopsychosocial variables on movement variability.

Dear Mr Shawn Robbins

We are pleased to inform you that the above-mentioned protocol has been granted institutional authorization for the Integrated Health & Social Services University Network of West-Central-Montreal, and / or under its auspices.

To grant this Institutional Authorization, it is understood that our establishments recognizes the approval granted by the reviewing REC.

- Whereby it is acting as the Reviewing REC for this project, under the MSSS Multicenter Mechanism "cadre de référence" for all public entities of the Health and Social Service Network;
- Affirming in their approval letter dated 2019-08-12 the positive outcome of the Science Review Committee and the Research Ethics Review Committee; and
- Affirms the approval of the consents forms required for the conduct of the study, inclusively, the French version of the consents forms.

We acknowledge receipt of the site-specific consent form(s) which include our Institutional requirements/modifications for the conduct of research with in the auspices of the West-Central Montreal Health and will attach copy to this Institutional Authorization that will be forwarded to Reviewing REC.

The documents submitted for institutional authorization as approved by the Reviewing REC have been granted institutional authorization. Please note that should at any point the Reviewing REC revoke, modify or change the status of your approval for Research Ethics, the Person Formally Mandated by the CODIM, retains the rights to revoke its authorization for the above-mentioned protocol.

This authorization also requires that you respect the terms and conditions listed below:

- Comply with the provisions of the "cadre de référence" MSSSS Multicenter Framework;
- Comply with the Regulatory Framework of West-Central Montreal Health with regards to research activities, including the requirements for the respect and privacy of research participants;
- Use only the documents and their respective versions that have been included in the approval by the Reviewing REC, with only possible changes including the administrative changes required by the West-Central Montreal Health – Office of the Person Formally Mandated to Authorize Research;
- Respect the mechanisms required for annual review determined by the Reviewing REC;
- Respect the procedures of the MSSS Multicenter Mechanism with regards to respect and privacy of research participants specifically, the identification of the research participants at our institution, that is maintaining the list of the participants

recruited into the study at our institution. This list must be submitted to the Office of the Person Formally Mandated to Authorize Research upon request;

- To ensure regulatory requirements of research files for the duration set by applicable regulations or the Reviewing REC, in the case of an audit; and
- To notify the Reviewing REC and Person Formally Mandated the ongoing conduct of the project, with regards to any modification to the research.

This authorization may be suspended or terminated by our Institution, in cases found to be non-compliant to the above-listed terms and conditions. The Reviewing REC will thus be notified.

It is understood that our Institution will communicate with the authorities with regards to personal information considered identifiable under the law, should there be, during the conduct of this study, proven cases of breaches of rules for the responsible conduct of research.

Your request for "Renewal of Institutional Authorization" must be received by the Office of the Person Formally Mandated to Authorize Research within 30 days of receipt of continuing review by the Reviewing REC of the above-mentioned study. Otherwise, the study's Institutional Authorization will be terminated and permission to conduct any research related activities within our CIUSSS will be prohibited. If any modification(s) to the study occurs (i.e., amendment) over the next twelve months, or should this study be completed during this period, please inform the Office of the Person Formally Mandated.

This authorization hereby grants you to perform research under the auspices of our institution and must be prior to the date specified by the Reviewing REC decision to renew its research ethics approval of this research and our Institution.

We trust this meets with your complete satisfaction.

Respectfully,

Cindy Starnino
Directrice des Affaires académiques | Director of Academic Affairs
Personne mandatée par l'établissement pour autoriser la réalisation des projets de recherche
CIUSSS du Centre-Ouest-de-l'Île-de-Montréal | CIUSSS West-Central Montreal

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