The use of technology in measuring low back function and morphology in low back pain patients
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Chapter 7

General discussion
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General discussion

About 40% of patients with “non-specific low back pain” (nLBP) do not completely recover within six weeks. Their pain becomes persistent (chronic or recurrent), regardless of whether the patients have received an intervention.\(^1\)\(^,\)\(^2\) It is expected that the number of patients with recurrent episodes of nLBP will increase, which will likely result in a concurrent increase in the demand for physiotherapy treatments.\(^3\)\(^,\)\(^4\) However, the outcomes of the majority of treatments, including, for example, stabilization training (i.e., lumbar multifidus [LM] training),\(^5\)\(^,\)\(^6\) and function-directed training (general exercise therapy), are positive.\(^7\) The results of systematic reviews comparing different treatment modalities for low back pain do not indicate that one approach can be favored over the other on a group level,\(^4\)\(^,\)\(^8\)\(^-\)\(^10\) but it is unclear if specific subgroups exist within this population that would favor the one approach over the other. This is partly because nLBP is considered a multifactorial problem, where psychosocial factors (such as an increased fear of movement and distress)\(^11\)\(^,\)\(^12\) and biological factors (such as low back muscle thickness or range of motion)\(^13\)\(^,\)\(^14\) most likely play an important role. Additionally, it is unclear what the role is of biological components within a disorder with associated biopsychosocial characteristics such as nLBP. Studies looking into the role of biological components appear to support the assumption that the LM muscle is responsible for the most substantial (about 70 percent) influence on functional spine stabilization.\(^15\)

However, with significant heterogeneity in patient samples, research methodology, and results, the role of the LM in patients with nLBP is unclear as a result of controversial evidence until now. Due to this controversial evidence, the clinical implications for treating patients with nLBP are limited for a physiotherapist. Therefore, the primary aim of the research described in this thesis is to increase the understanding of the role of the LM in patients with nLBP.

This last chapter will start with a summary of the main results from this thesis, after which the results will be placed in a broader context. Subsequently, the clinical relevance and the implications for the clinical scenario described in the general introduction will be discussed. Finally, recommendations for future research and the overall conclusions will be presented.

Main thesis findings

We studied the contribution of the LM in the low back in both healthy people and patients with nLBP using a literature review and a series of experimental studies. To gain a deeper understanding of the assumed amount of anatomical heterogeneity in LM research, the literature was systematically reviewed for patient samples and research methodology. Our results indicate that several previously reported research methods measuring LM morphology and low back function are not reproducible, and their validity can be questioned. The results show that images of LM morphology have
a large variety in depiction, in description in literature, and in anatomy atlases. The included anatomical studies show inconsistent findings, describing the location of the LM as superficial (50%), deep (25%), or superficial and deep (12%) in reference to the skin. In addition, the most cited literature supports that the LM is a superficial muscle at the spinal levels from S2 till L3-4 (Chapter 2), and that it is recommended to perform more research addressing standardization of the LM anatomy and LM ultrasonography as a prerequisite for evidence-based physiotherapy.

This led to our aim to develop a guide for physiotherapists to better elucidate the anatomy of the LM in Chapter 3. For that purpose, we photographed deep-frozen human tissue of lumbar spines in 3D blocks, which enabled us to reconstruct cross-section photographs that matched ultrasound imaging obtained from a healthy volunteer. We revealed new insights into the topography of LM with 3D modelling and ultrasound. This provided a more detailed anatomy, improving the understanding of standard LM ultrasound imaging, which resulted in a more adequate ultrasound measurement of the borders of the LM compared to adjacent musculature. The results also show that the maximum muscle thickness of the LM in the transversal plane is at the level of L5-S1. With this result, we studied the positioning of the surface electromyography (EMG) and specifically, how the electrode position matched the current guidelines for electrode positioning in EMG. This is standardized by the European Guideline “Placement Surface EMG for the Non-Invasive Assessment of Muscles (SENIAM),”16 which dictates placing the surface EMG electrode to assess the LM at the level of L5. Consequently, we concluded, based on our anatomical study, that the positioning of the surface EMG electrodes should be refined to more accurately measure the activity of the LM (also by using ultrasound).

Subsequently, in Chapter 4, we conducted a study to examine our hypothesis of electrode positioning as described in Chapter 3. In an experimental setting with 15 healthy adults, we compared two methods of surface EMG with the current gold standard of intramuscular EMG to quantify the amount of true signaling from the LM compared to the amount of cross-talking and co-contraction. The healthy adults performed several clinical tests aimed at creating (isolated) LM contraction. In all these clinical tests, the muscle activity of the LM and erector spinae (ES) measured with surface EMG correlated well to the LM and ES activity measured by intramuscular EMG (true signals). However, significant cross-talk and co-contractions were also observed in the combinations with the surface and intramuscular EMG electrodes of LM and ES. Such high correlations of cross-talk and co-contraction of the LM and ES muscles demonstrate that both muscles are concurrently active in low back movements. Based on this study, we concluded that the EMG measurements of the LM and ES indicate that none of the applied clinical tests appear to be able to activate isolated LM activity (Chapter 4). In other words, the LM and ES are simultaneously active in all clinical tests (and likely also in activities in daily life [ADL]) in healthy people. Viewed together, these results indicate that the LM and ES both contribute to muscle synergy for low back movements.
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In chapters 5 and 6, we aimed to bring measurements such as, muscle activity, muscle thickness, and trunk Range of Motion (ROM), into physiotherapy practice in a large cross-sectional study to test whether technologies (EMG, ultrasound, and 3D kinematics) can be used to identify abnormal or deviating patterns of low back muscle in patients with nLBP compared to healthy subjects. A total of 161 subjects, 52 patients with subacute nLBP, 59 patients with chronic nLBP, and 50 healthy controls, were included. In Chapter 5, we looked for differences in LM morphology and low back function between the groups of patients with (subacute and chronic) nLBP and the healthy controls. An important finding is that trunk ROM and LM thickness were lower in patients with nLBP compared to the healthy controls (Chapter 5).

In Chapter 6, we studied the associations between disability, pain, and biopsychosocial factors in patients with subacute and chronic nLBP. The results indicate that disability is associated with the LM characteristics of LM thickness, trunk ROM, EMG of the LM, and specific patient-reported outcome measures, such as fear-avoidance, beliefs about work, and assumptions for central sensitization. Pain intensity, however, was to a far lesser extent associated with the LM characteristics in patients with nLBP. In addition to the aim of identifying the associations between disability, pain, and biopsychosocial factors, we tested differences in LM morphology and low back function between the groups of patients with subacute nLBP and patients with chronic nLBP. The results show that LM thickness is lower in patients with chronic nLBP compared to patients with subacute nLBP. Overall, these findings demonstrate that LM thickness and trunk ROM, as biological components, are involved in nLBP, including chronic nLBP.

**Muscle synergies in low back movements**

The new perspectives on the LM anatomy presented in Chapter 3 set the basis for further validation studies on the use of technology for the EMG measurements of the LM muscle in daily practice. We expected that the alternative surface EMG electrodes positioned on the LM would show less cross-talk and co-contraction compared to the surface EMG electrodes positioned on the LM, based on the European Guideline Placement SENIAM.16 Remarkably, this was not the case. No substantially less cross-talk was measured between these two different surface EMG electrode positions on the LM. This means that in both surface EMG electrode positions, the LM muscle activity cannot be measured solely, without measuring adjacent muscle activity. Our experiments indicated that this could be due to insufficient discriminatory capacity of the surface EMG for the LM or that no isolated LM activity can be performed; most likely, both elements play a role. In addition, our clinical experiment in Chapters 5 and 6 shows no differences between patients with subacute and chronic nLBP and healthy controls in muscle fatigue by surface EMG measurements of the LM. These EMG results support that surface EMG measurements of the LM might have an insufficient discriminatory capacity for measuring individual low back muscles.
The finding of co-contraction in all clinical tests in our EMG results is reason to consider the presence or influence of low back muscle synergies. A muscle synergy is a group of two or more muscles that are linked together by spinal neural circuitry. When the circuitry is activated, the group of muscles contracts and/or relaxes in a characteristic, coordinated pattern. The LM and ES muscles could be part of a low back muscle synergy. Perhaps when more low back muscles are measured simultaneously with (surface) EMG, low back muscle synergies could be measured. Differences between healthy controls and patients with nLBP have been found at muscle activation level to achieve low back motion. This difference at muscle activation level might imply that for low back motor control, healthy controls might sooner activate a muscle synergy in low back motion compared to patients with nLBP. There may thus be a relation between low back muscle synergies and the duration of the nLBP disorder. In patients with chronic nLBP, where the disorder is present longer than in other patients with nLBP, the disorder duration might influence inadequate trunk muscle recruitment patterns. An alternative hypothesis is that patients with nLBP might use an altered trunk muscle activation strategy to minimize the pain. These considerations indicate that clinicians should focus more on muscle activation patterns in low back movement than on the isolated activation of one muscle.

**Trunk range of motion (ROM) as a discriminator**

Next to the technology of EMG, 3D kinematics were also measured with a 3D sensor with the outcome of trunk ROM. This thesis shows that trunk ROM is a discriminating measurement between patients with nLBP and healthy subjects (Chapter 5). It supports the theory that differences between patients with nLBP and healthy subjects are shown when a movement is made through which low back muscle synergies are used. Patients with subacute and chronic nLBP show less ROM in various trunk movements (flexion, extension, and lateral flexion) than healthy controls, but we could not identify differences between chronic and subacute patients. This may suggest that this specific body function is affected relatively early in the process of low back pain. For future research, this requires longitudinal study designs to measure trunk movement over time in patients with nLBP. Trunk ROM was also one of the associated factors of disability in patients with nLBP. However, further research is recommended to examine whether this reduced ROM has a causal relationship to disability in patients with nLBP (Chapter 6). It should be mentioned that trunk ROM not only expresses the LM muscle function specifically, but also better supports the theory of muscle synergies. Our trunk ROM tests are an expression of the low back function’s active movement, instead of one segment’s active movement or only LM muscle function.

On the other hand, physiotherapists experienced the 3D sensor as an easy tool to monitor low back movements in clinical practice. The tool is delivered with easy-to-install software. It has great versatility and small dimensions, and the high mobility makes it
possible to carry out tests almost anywhere. All in all, trunk ROM is a relevant factor for monitoring and evaluating low back function in an individual patient with nLBP. Trunk ROM measurement with a 3D sensor is a clinically applicable tool in primary care physiotherapy.

The role of the LM in patients with nLBP

Chronic nLBP is widely recognized as a multifactorial disorder. The main results of Chapter 5 indicate that the patients with nLBP studied have less trunk ROM and lower LM thickness than the healthy controls, which implies that there is an association between LM and nLBP. It was expected that trunk ROM, LM muscle fatigue, and LM thickness (all three LM characteristics) were associated with disability in patients with nLBP. This result indicates that in patients with nLBP a lower LM thickness, lower trunk ROM, or more LM muscle fatigue is associated with a higher level of disability. The literature supports the results of Chapter 6 that LM morphology is associated with disability. In addition, Ranger et al. (2019) support a relationship between a thinner lumbar paraspinal muscle (LM and ES muscles) and higher levels of disability. This source suggests that treatment strategies directed at increasing paraspinal muscle size may be effective in reducing low back disability in patients with chronic nLBP. So, this evidence supports a reappraisal of the biological component, especially the LM muscle, within a biopsychosocial model in approaches for nLBP in primary care physiotherapy.

Clinical relevance

In the following section, we will discuss the implications of our findings for training methods to treat nLBP and the use of the technology in primary care physiotherapy. Subsequently, we will discuss the outcomes of the clinical scenario from the general introduction.

In physiotherapy, there are two “main” streams of exercise therapy treatments for nLBP. Both treatments, i.e., stabilization training (for example, LM training) and functional training (for example, general exercise therapy, cognitive functional training, graded activity, and graded exposure training) show positive outcomes. Both therapies may include strengthening exercises for the lower back. These strengthening exercises aim to improve the trunk muscle volume that is expected to restore the strength of the trunk muscles, which might improve coordination and control of the trunk muscles of the lumbar spine as well. The main difference between these methods is that functional training is more focused on behavioral aspects than stabilization training, which aims to achieve physical improvements, particularly through LM muscle strengthening training.
Evidence for stabilization training

We found some support for stabilization training in patients with nLBP. Our results indicate an association with LM, including LM thickness, and patients with nLBP. For example, LM thickness is lower in patients with nLBP compared to healthy people, and LM thickness is inversely proportionally associated with disability and, to a lesser extent, to pain intensity in patients (Chapters 5 and 6). This indicates that LM thickness could be a sustaining factor or a consequence of nLBP, including chronic nLBP. Results also suggest that training programs that focus on the increase of LM muscle volume or thickness, i.e., LM training, can be effective in reducing low back disability and pain intensity in patients with nLBP. Training that distinguishes between deep and superficial muscle training appears to be outdated because we could not verify a clear identification of deep versus superficial LM (Chapter 3). The literature shows that low back muscle size can indeed increase and disability can decrease in a stabilization training program (with motor control exercises).

Evidence for functional training

Most of our results imply that functional training treatments should be preferred for patients with nLBP. In our clinical tests, the LM muscle could not be activated solely; we always observed concurrent activation of the ES muscle (Chapter 4). This indicates that the LM and ES might both be active in low back muscle synergies. Therefore, it appears impossible to train the LM muscle individually through an exercise. This implies that strengthening exercises will focus on the improvement of the trunk and low back muscle mass instead of only the LM muscle mass. Moreover, Chapter 6 indicates that disability is more strongly associated with trunk ROM, LM muscle fatigue, LM thickness, and patient-reported outcomes (such as central sensitization and fear-avoidance beliefs at work) than with pain intensity. This implies that more factors (e.g., fear-avoidance beliefs and distress) than only the biological factors of the LM are associated with nLBP. The literature supports the recommendation of exercise programs that target functional improvement and prevention of increasing disability. Furthermore, guidelines from Denmark, the United States, the United Kingdom, and the Netherlands recommend exercise programs that consider individual needs, preferences, and capabilities when selecting an exercise type. The literature and our findings both support that functional training is more favorable than stabilization training for patients with nLBP. While both approaches appear non-inferior on effect, we can conclude with reasonable evidence that the treatment effects are equal, but the working mechanism is better described in functional training methods. When it comes to clinical relevance, it is essential to explain to patients that low back pain is a multifactorial syndrome in which biological, psychological, and social factors interact closely and that gaining control of low back musculature alone is not going to solve their problem.
Use of technology in primary care physiotherapy

Our results (Chapters 3 and 4) indicate that the LM muscle is a superficial muscle and can be measured well using ultrasound in primary care physiotherapy practices. From the clinical perspective, LM thickness measured via ultrasound and trunk ROM measured via 3D sensor can be used for individual monitoring in patients during treatment in primary care physiotherapy, as independent and objective outcome measures that are related to the disability of patients, as a valuable additive to psychosocial questionnaires.

Our experiments show that the surface EMG of LM measures adjacent muscle activity without isolated LM activity. We have no clinical tests or low back movement showing that solely activates the LM. Our physiotherapists examined the raw EMG data directly on the screen during the tests. However, it was hard to interpret the data directly after the measurement. Based on these results, more research should be done to explore monitoring low back muscle activity as an outcome in patients with nLBP in primary care physiotherapy. In addition, an EMG measurement protocol should be developed for physiotherapists in primary care, determining how and when to monitor low back muscle activity in patients with nLBP to develop better clinical practice in physiotherapy.

Clinical scenario from the general introduction

It is important to consider the clinical relevance of our findings for professionals in daily physiotherapy practice. This thesis started with a general introduction describing a clinical scenario of a 39-year-old woman who had been experiencing LBP for 4 weeks. Physiotherapist Anne proposed to start stabilization training and to monitor LM muscle thickness with ultrasound. Physiotherapist Tukker proposed functional training and to monitor low back muscle activity with EMG. Based on which findings and measurement tools should these physiotherapists choose which training method and monitoring tool to apply in this clinical scenario? Based on our findings, physiotherapist Tukker’s choice of functional training would be more suitable for his patient with nLBP than Anne’s choice of stabilization training. We consistently observed concurrent activation of the ES muscle and LM muscle. In addition, we have implied that more factors (e.g., fear-avoidance beliefs and distress) than only the biological factors of LM are associated with nLBP. All these factors would be trained in functional training and not in stabilization training.

For monitoring, physiotherapist Anne chose the most valid measurement tool. LM thickness is a factor that showed differences between healthy subjects and patients with subacute and chronic nLBP, and between patients with subacute and chronic nLBP; this factor is associated with disability. In addition, LM thickness can be measured well with ultrasound by a physiotherapist in primary care. Surface EMG of low back muscles indicated cross-talk and co-contraction in reported LM and ES EMG signals.
Additionally, surface EMG measurements of the low back muscles did not differ between healthy subjects and patients with subacute and chronic nLBP, nor between patients with subacute and chronic nLBP. We advise monitoring LM muscle thickness with ultrasound rather than low back muscle activity with surface EMG in primary care physiotherapy. Overall, functional training as the training option (the choice of physiotherapist Tukker) and the ultrasound to monitor LM thickness as the technology option (the choice of physiotherapist Anne) is the best combination for our clinical scenario.

**Recommendations for future studies**

Besides the clinical relevance, the results of this thesis also inspire new research ideas. We found that trunk ROM and LM thickness were lower in patients with nLBP compared to healthy controls and that disability is associated with the biological factors of LM thickness, trunk ROM, and EMG of LM in patients with nLBP. Based on these results, we cannot state whether biological factors are predictors for chronic nLBP or how we could identify or treat patients at risk for developing nLBP. Currently, no norm values are available to compare biological factors such as LM thickness in healthy people and in patients with acute, subacute, or chronic LBP. These norm values could help a clinician in primary care practice improve the diagnosis of (n)LBP and/or monitor these patients for LM thickness. If these norm values were to be established, LM thickness measured via ultrasound could be an outcome measure to add to a clinical prediction rule of (n)LBP.37

In this thesis, the focus was principally on the function and morphology of the LM muscle. However, the ES muscle might also play an essential role in low back movement and the development of subacute or chronic nLBP. Therefore, we recommend investigating the validity of the surface electrode placements for the low back muscles, measuring each low back muscle individually and integrating this information in the EMG guidelines, especially for physiotherapists in primary care.

Overall, a longitudinal study is recommended to identify predictive characteristics for the transition from acute, to subacute, to chronic nLBP compared to patients with acute nLBP who do not develop chronic nLBP. Biological factors, as well as psychosocial factors, should be included as outcome measures in such a study. In an ideal scenario, these outcome measures should be measured in a clinical setting, to increase the generalizability to the primary care setting of physiotherapy. More knowledge about these predictive factors is needed to tighten up the current guidelines and clinical prediction rule for nLBP.
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Concluding remarks

The studies in this thesis have increased our understanding of LM anatomy description, LM morphology and function, and low back function in healthy individuals and patients with nLBP. We conclude that the LM is a superficial muscle from S2 until L3-4, with a maximum muscle thickness at the level of L5-S1, which can be monitored well with ultrasound in primary care physiotherapy. The surface EMG measurements of LM might have insufficient discriminatory capacity for measuring individual low back muscles. LM thickness and trunk ROM, as biological factors, are also involved in nLBP, including chronic nLBP. nLBP is a multifactorial problem. Psychosocial and biological factors play an essential role, and our studies included a vital set of biological factors. However, this research is just the beginning; the tip of the iceberg. In future research, we must focus on these biological factors in the current multifactorial approach in diagnostics and training methods for patients with nLBP in primary care.
References

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