Summary

Chronic non-specific low-back pain (LBP) is a common health problem in Western society, affecting a large majority of the population. Many patients recover fairly quickly without specific treatment, but relapses are common and for a large minority, LBP develops into a chronic problem. Many risk factors have been identified, but the evidence for the causal relationship between these factors and LBP is weak. Common treatments often focus on one or several prognostic factors (neuromuscular control, pain sensitization and pain-related fear) and there is some evidence that exercise therapy targeting neuromuscular control is effective. However, a large number of patients does not respond to treatment and this limited success is often attributed to a lack of adequate diagnostics. The changes in neuromuscular control with LBP are diverse and complex with evidence of both increased and decreased excitability. These changes may cause pain and pain recurrence, due to e.g. tonic muscle activity, but may also be protective against pain and re-injury by stabilizing the spine. Gaining further insight in the neuromuscular control of the low back seems essential for a breakthrough in the current treatment methods of chronic LBP.

Low-back stabilization involves a complex biomechanical system that counteracts the downward pull of gravity on the large mass of the upper body while it balances on top of the lumbar vertebrae which, in turn, balance on the sacrum. The human spine is not structurally stable and the musculature is essential to prevent the spine from buckling. Spinal stiffness can be provided by intrinsic components (passive tissues and agonist-antagonist muscle co-contraction) and reflexive components (muscle activation initiated by feedback from sensory organs). How these components interact and contribute to low-back stabilization is still unknown.

The goal of this thesis was to advance the understanding of the neuromuscular control in low-back stabilization and to gain insight into the interaction between low-back stabilization and low-back pain. To achieve this goal, three main research questions were formulated:

1. Can the intrinsic and reflexive contributions to low-back stabilization be determined reliably?
2. How does low-back stabilization modulate between different conditions and task instructions?
3. How does low-back stabilization differ between healthy subjects and LBP patients?

Since the experimental methods applied throughout this thesis imply that the subjects is in contact with an external object (the pushing-rod applying the external perturbation), the second part of this thesis deals with the following additional research questions:

1. Does tactile information on the back interact with sensory feedback from other sources (i.e. does it lead to sensory reweighting)?
2. Does sensory reweighting occur with a moving source of tactile information?
3. Does tactile information interact with sensory feedback even when the source of tactile information is moving in an unpredictable manner?

To answer these questions, new experimental protocols had to be developed.

**Developing the method**

In chapter 2, a systematic review into methods used to assess trunk stabilization showed that many different methods exist but that not all measure the contributions of co-contraction and reflexes simultaneously, which may pose a threat to the validity of the results and might lead to misinterpretations. Therefore, in chapters 3 and 4, we set out to develop a method that can distinguish between intrinsic and reflexive contributions to low-back stabilization and demonstrated good test-retest reliability.

**Modulation of low-back stabilization**

Substantial modulation of low-back stabilization was found due to task instruction (chapter 3) and posture (chapter 5). Compared to a natural low-back stabilization task, the instruction to maximally resist the perturbation led to decreased lumbar movement. This was achieved by increased co-contraction and velocity feedback. While an extended lumbar posture did not significantly change low-back stabilization, lumbar flexion resulted in higher low-back resistance, reduced reflexive contributions and lower co-contraction levels. The flexion-relaxation phenomenon, i.e. reduced muscle activity in a maximally flexed posture due to increased passive tissue stiffness, may explain this result.
**Low-back stabilization with low-back pain**

Low-back stabilization was compared between healthy controls and LBP patients during trunk perturbations, while either maximally resisting the perturbation or stabilizing the low-back in a natural way (chapter 6). Compared to the control group, the patients displayed less reflexive modulation due to task instruction and higher intrinsic contributions during maximal stabilization, suggesting impaired reflexive adaptation in LBP. In line with literature, this thesis describes diverse changes in motor control with LBP, where individual patients showed either an increase or decrease in admittance, reflexes and/or modulation, indicative of heterogeneity within the LBP patient group. This suggests that sub-populations of LBP patients may show different and even opposite changes in motor control, indicating clinically relevant sub-groups.

A new categorization of LBP patients was proposed based on the maximal low-back stabilizing ability (task instruction to resist the perturbation) and the modulation towards natural low-back stabilization. Tentatively, four sub-groups of patients were defined, each with an unique pattern of motor control differences relative to healthy controls: no low-back motor control impairment (1), low-back muscle weakness (2), limiting low-back muscle forces (3) and limiting low-back movements (4).

**Tactile information in low-back stabilization**

In the second part of this thesis, the influence of tactile information on trunk control was investigated. First, in chapter 7, it was shown that tactile information through hand and back interacts with the contribution of other sensory modalities (vestibular and proprioceptive). In chapter 8, it was shown that the source of tactile information does not have to be stationary and that the sway of the upper body becomes entrained to the motion of the tactile source. Finally, in chapter 9, it was shown that the interaction effect between tactile information and other sensory modalities still holds when the source of tactile information is moving in an unpredictable way. Therefore, when applying external perturbations, the tactile information provided by contact with the perturbation apparatus should be considered a significant contributor to sensory feedback.