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DISSERTATION TITLE: *Lumbar radicular pain and disc herniation: Genetic factors, pathophysiological mechanisms and pain assessments*

In study 1 and 2 of the thesis, we addressed the mechanisms underlying development of persistent pain in patients with lumbar radicular pain after disc herniation. The results showed that subjective health complaints in this patient population were associated with a genetic variability in the gene encoding the opioid receptor OPRM1 (μ-opioid receptor 1 polymorphism A118G). Women with the OPRM1 A-variant reported more subjective health complaints than men with the same genotype. In the same patients, the pain-intensity was also associated with the serum level of microRNA-17. Subsequent laboratory work demonstrated an up-regulation of microRNA-17 in herniated rat intervertebral disc tissue, and increased release of TNF (an inflammatory signalling protein) when microRNA-17 was transfected into monocytic immune cells (a THP-1 cell line).

In study 3 and 4 of the thesis, we further evaluated two widely used questionnaires for patients with pain. In the patient population described above with lumbar radicular pain after disc herniation, we found that the ability of the painDETECT questionnaire to detect neuropathic pain was poor when compared against a clinical reference standard for neuropathic pain. Thus, our results did not support the use of painDETECT as a screening tool to classify neuropathic pain in patients with lumbar radicular pain. Moreover, we performed a translation of the Short-Form McGill Pain Questionnaire-2. The Norwegian questionnaire showed excellent acceptability and comprehension by patients and had satisfactory measurement properties. However, our analysis raised concerns regarding the earlier established four-factor structure. Until more evidence emerges, we suggest using this questionnaire as a single measure.