teomes for each patient were relatively quantified using a mass spectrometry based shotgun approach.

Results: In total, 419 unique proteins were simultaneously identified and relatively quantified. A panel consisting of seven proteins, 5 up-regulated and 2 down, were found to be significantly regulated by SCS in two complementary statistical tests ($P \leq 0.01$). The most up-regulated protein in the SCS linked panel is a known modulator of nicotinic acetylcholine (ACh) receptor activity. Interestingly, it has a striking tertiary structural similarity and biological functionality as pain modulating prototoxins found in snake venoms.

Conclusions: Our findings reveal possible insights into the mechanism of spinal cord stimulation and the obtained pain relief.

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B3

The DQB1(∗)03:02 HLA haplotype is associated with increased risk of chronic pain after inguinal hernia surgery and lumbar disc herniation

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Abstract: Neuropathic pain conditions are common after nerve injuries and are suggested to be regulated in part by genetic factors. We have previously demonstrated a strong genetic influence of the rat major histocompatibility complex on development of neuropathic pain behavior after peripheral nerve injury. In order to study if the corresponding human leukocyte antigen complex (HLA) also influences susceptibility to pain, we performed an association study in patients that had undergone surgery for inguinal hernia ($n = 189$). One group had developed a chronic pain state following the surgical procedure, while the control group had undergone the same type of operation, without any persistent pain. HLA DRB1genotyping revealed a significantly increased proportion of patients in the pain group carrying DRB1(∗)04 compared to patients in the pain-free group. Additional typing of the DQB1 gene further strengthened the association; carriers of the DQB1(∗)03:02 allele together with DRB1(∗)04 displayed an increased risk of postsurgery pain with an odds risk of 3.16 (1.61–6.22) compared to noncarriers. This finding was subsequently replicated in the clinical material of patients with lumbar disc herniation ($n = 258$), where carriers of the DQB1(∗)03:02 allele displayed a slower recovery and increased pain. In conclusion, we here for the first time demonstrate that there is an HLA-dependent risk of developing pain after surgery or lumbar disc herniation; mediated by the DRB1(∗)04–DQB1(∗)03:02 haplotype. Further experimental and clinical studies are needed to fine-map the HLA effect and to address underlying mechanisms.

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B4

On the pharmacological effects of two lidocaine concentrations tested on spontaneous and evoked pain in human painful neuroma: A new clinical model of neuropathic pain

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Aims: Spontaneous and evoked pains are key symptoms of patients with neuropathic pain and there is a current discussion on the predictive value of evoked pain read outs for the reduction of spontaneous pain. Here we describe a new clinical model of neuropathic pain that may be useful for evaluation of new drugs. We also report the effects of lidocaine in this model as reference values.

Methods: In a randomized double-blind experiment, the analgesic effects of local lidocaine were investigated separately for spontaneous pain and for stimulus-evoked allodynia and hyperalgesia in sixteen patients with painful neuromas after traumatic nerve injuries in the upper extremities. The patterns of sensory changes were compared before and after treatment with lidocaine (0.1% or 0.5%, 1 ml), with 1–2 weeks interval, injected close to the neuroma. Spontaneous and evoked pains were assessed using a visual analogue scale (VAS) and quantitative/qualitative sensory testing.

Results: Lidocaine dose-dependently reduced spontaneous and evoked pain scores by more than 90% with maximum effects between 1 and 5 min for evoked pain and between 3 and 15 min for spontaneous pain. While evoked pain normalized rapidly reaching about 50% of the control level 20 minutes after the injection spontaneous pain levels were lower than 25% at this time. Moreover, in 4 patients the reduction of ongoing pain lasted 24 h whereas evoked pain had returned to baseline levels in all the patients after 1 h.

Conclusion: Differential analgesic effects of local lidocaine on spontaneous and evoked pain suggest that different mechanism underlie these two key clinical symptoms. Thus, clinical trials assessing localized traumatic neuropathic pain should investigate both aspects of pain separately with the proposed model allowing testing of new drugs systemically or locally administered.

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