the joints, synovial neovascularization and proliferation of fibroblasts and bone erosion. Clinically the disease is evident as joint inflammation, swelling and progressive joint destruction. Of importance, pain is one of the most bothersome symptoms reported by RA patients. Autoantibodies to type II collagen (CII) and glucose-6-phosphate isomerase (GPI) are detected in serum of 70% and 20% of the RA patients, respectively. Intraperitoneal injection of these antibodies to mice rapidly induces arthritis-like symptoms and generates a joint pathology that resembles human RA. While collagen antibody-induced arthritis (CAIA) and K/BxN serum transfer (GPI antibody-mediated) are common models in the rheumatology field, they have not been evaluated as models of arthritis-induced pain. Data from our studies in which pain behavior (tactile and cold allodynia) and the analgesic effect of ketorolac (cyclooxygenase inhibitor), etanercept (TNF inhibitor) and gabapentin were examined will be presented. In brief, we observed that both injection of CII and GPI antibodies caused induction of clinical signs of arthritis including joint swelling and redness of the paws, and that this inflammatory state gave rise to a robust, and reproducible allodynia. Interestingly, the allodynia outlasted the signs of joint inflammation. Of note, while intraperitoneal injection of ketorolac, etanercept and gapapentin attenuated arthritis-induced allodynia during the inflammatory phase, only gabapentin had anti-allodynic effect in the "post-inflammatory" phase. Spinal activation of astrocytes and microglia was assessed as these cells have been implicated to play a role in the maintenance of hypersensitivity in experimental models of persistent pain. Experiments in which microglia and astrocyte activity was assessed using quantitative real time PCR and immunohistochemistry indicated activation of spinal glia in both models.

In summary, our work demonstrates that the CAIA and K/BxN arthritis models generate robust and highly reproducible allodynia. Based on the resemblance with human pathology these models may become important assets in dissecting the mechanisms that drive arthritis-induced pain, both during peak and remittent phases of the disease.

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Translating basic research to pharmacological treatment of neuropathic pain

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Despite the major advances in our knowledge of mechanisms involved in neuropathic pain, patients continue to suffer from chronic and disabling neuropathic pain conditions. Over 150 randomized controlled studies have evaluated the effect of pharmacological agents for neuropathic pain conditions, including postherpetic neuralgia, painful polyneuropathy, peripheral nerve injury, and central pain. Despite an increase of about 60% in new randomized placebo-controlled trials in neuropathic pain during the past 5 years, there seems to be no evidence for major changes of the treatment algorithm. Tricyclic antidepressants (TCAs), serotonin noradrenaline reuptake inhibitors (SNRIs), the anticonvulsants gabapentin and pregabalin, and opioids are the drug classes with most consistent efficacy. Topical treatments with lidocaine patch, botulinum toxin A, and high-dose capsaicin are new treatment modalities which seem to have some effect on peripheral neuropathic pain with little or no systemic side effects. Other drug classes seem to relieve neuropathic pain in subgroup of patients, but more trials are needed to evaluate the overall effect and predictors of efficacy of these drugs, which include lamotrigine, oxcarbazepine, carbamazepine, valproate, and lacosamide. Certain drugs do not relieve neuropathic pain despite promising results from basic research. These drugs include NK_1-receptor antagonists and the anticonvulsant levetiracetam.

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Free Poster Presentations

Forearm heat pain does not inhibit electrically induced tibialis anterior muscle pain

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Objectives: Women report more musculoskeletal pain than men. A dysfunctional pain inhibitory system has been launched as a contributing factor for these gender differences. This study used a diffuse noxious inhibitory controls (DNIC) paradigm and asked the following questions: (1) is electrically induced muscle pain inhibited by a painful heat stimulus to the forearm, and (2) does women show signs of reduced inhibition compared to men?
Methods: Forty healthy subjects (20 females; 18–45 years) participated in a cross-over design with painful (45–49 °C) or non-painful (35 °C) conditioning heat stimuli (in balanced order) to the contralateral forearm. The subjects received 10 painful electrical stimuli in the tibialis anterior muscle before, during and after conditioning and rated each electrical stimulus on a 0–10 cm visual analogue scale (VAS). There was 30 min between experiments. All VAS scores were normalized to scores before conditioning (100%) and analyzed by RM-ANOVA. Females participated during the ovulatory phase (days 12–14).
Results: There was a main effect of conditioning. VAS scores during conditioning were reduced to $90 \pm 24\%$ (mean ± S.D.) with respect to before conditioning ($p = 0.02$). There was no difference between painful and non-painful conditioning ($p = 0.31$). Neither was there any difference between genders ($p = 0.28$); mean male VAS scores were $91 \pm 21\%$ and mean female scores were $89 \pm 27\%$ during (vs. before) conditioning.
Conclusion: Electrically induced muscle pain was not inhibited by a painful heat stimulus to the forearm. The reduction in muscle pain may be results of habituation. The lack of a DNIC effect may have been caused by the relatively small skin area receiving the conditioning stimulus, and excludes a conclusion on gender effects.

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Offset analgesia evoked by non-contact thermal stimulator

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Objective: The objective of this study was to test if offset analgesia could be evoked using a noncontact thermal stimulator. Offset analgesia [J. Neurophysiol. 87:2205–2208, 2002] is defined as an unproportionally large decrease in pain intensity following a slight decrease in stimulation intensity. The importance of differences in thermal properties between human hairy and glabrous skin was investigated.
Methods: A 20W diode laser (970 nm) was used for the thermal stimulation. A fast (50 images/s) infrared camera measured the skin temperature and a temperature controlled feedback control