presents study was to explore the nervous system by assessment of esophageal sensitivity to multimodal stimulations.

Methods: Throughout an euglycemic clamp, 31 healthy volunteers (age 44.3 ± 10.6 (mean ± SD) years; 11men) and 31 patients (age 46.3 ± 11.7 years; 10 men) with insulin dependent diabetes mellitus (duration 31.3 ± 13.1 years) were included in the study. By use of a multimodal oesophageal probe, sensitivity to heat, mechanical distension and electrical stimulation was assessed in the lower oesophagus.

Results: For heat stimulation patients had increased sensitivity in the sensory range with shorter stimulus duration until pain tolerance threshold (122 ± 3.8 sec vs. 136 ± 3.7 sec; $p = 0.006$) and larger area under the temperature curve (2380 ± 1847 vs. 1409 ± 1450; $p = 0.03$). There were no differences between groups for mechanical stimulation (maximum pressure (39 ± 57 mmHg vs. 24 ± 48 mmHg; $p = 0.3$; maximum volume 59 ± 21 ml vs. 62 ± 25 ml; $p = 0.56$). As an overall finding, patients tolerated higher electrical stimulation intensities ($p = 0.02$), dominated by discrimination in the sensory range: At sensory detection threshold (VAS1) 21.1 ± 12.4 mA vs. 16.3 ± 5.5 mA ($p = 0.03$); at moderate sensation (VAS3) 27.5 ± 13.3 mA vs. 21.5 ± 5.0 mA ($p = 0.03$) however at pain detection threshold (VAS5) 31.6 ± 13.1 mA vs. 28.8 ± 5.2 mA; the trend was insignificant ($p = 0.3$).

Conclusion: Patients with insulin dependent diabetes mellitus had modality-specific alterations of esophageal sensitivity. Heat stimulation activates selectively mucosal receptors whereas on the contrary electrical stimulation depolarizes the free nerve endings non-selectively. Hence, due to the different sensitivity profile, the esophageal neuropathy is most likely a result of both peripheral and central neuropathy caused by longstanding diabetic neuronal damage.

http://dx.doi.org/10.1016/j.sjpain.2012.05.023

Validation of a porcine behavioural model of UVB induced inflammatory pain

Pierpaolo Di Giminiani [1,*], Lars J. Petersen [2,3], Mette S. Herskin [1]

[1] *Department of Animal Science, Aarhus University, Blichers Allé, DK 8830 Tjele, Denmark*
[2] *Department of Clinical Physiology, Viborg Hospital, Heibergs Alle 4, DK-8800, Viborg, Denmark*
[3] *Department of Health Sciences and Technology, Aalborg University, Fredrik Bajers Vej 7, DK-9220 Aalborg, Denmark*

Background and aim: Cutaneous inflammation induced by ultraviolet B-light (UV-B) is considered a valuable translational pain model. Until now, the development of primary hyperalgesia has been assessed predominantly in rodents, whereas porcine skin might be advantageous due to its greater homology with human skin.

The aim of the present study was to investigate porcine behavioural responses to nociceptive mechanical and thermal stimulations following UV-B inflammation.

Methods: One skin area of 4 cm^2 in the flank of 16 male pigs of 55 ± 6 kg was irradiated by UV-B using 3xMED (Minimum Erythema Dose).

Changes in pain sensitivity were assessed 24 and 48 h following irradiation via delivery of mechanical (Pressure Application Measurement device) and thermal (CO_2 laser) stimulations to the inflamed skin area and to an untreated control site.

Results: All animals showed higher sensitivity in the inflamed skin site 24 and 48 h following irradiation, compared to the control site ($P < 0.05$). Pressure withdrawal threshold decreased in the inflamed site: 231 g (148–451) against 408 g (347–684) after 24 hours and 200 g (106–293) against 656 g (405–902) after 48 h. Similarly, latency to respond to the laser stimulus was lower at the inflamed site: 5 s (3–7) against 9 s (4–2) after 24 h and 4 s (3–15) against 20 s (8–25) after 48 h.

One and two days after irradiation, a tendency was found for an increased cutaneous mechanical pain sensitivity compared to baseline values in the site irradiated with UV-B light ($P = 0.092$). Thermal sensitivity was increased within the inflamed site 24 h after irradiation with latency changing from 17 s (4–25) at baseline to 5 s (3–7) at 24 h ($P = 0.001$). At 48 h, the response latency had not decreased any further ($P = 0.414$).

Conclusions: Our study shows that behavioural recordings are a valid tool for the assessment of mechanical and heat sensitization following UV-B inflammation in porcine skin.

http://dx.doi.org/10.1016/j.sjpain.2012.05.024

Recovery after a lumbar disc herniation is dependent on a gender and OPRM1 Asn40Asp genotype interaction

M.B. Olsen [1,*], L.M. Jacobsen [1], E.I. Schistad [2], L.M. Pedersen [1,2], L.J. Rygh [3], C. Røe [2,4], J. Gjerstad [1,5]

[1] *National Institute of Occupational Health, Norway*
[2] *Department of Physical Medicine and Rehabilitation, Oslo University Hospital, Ullevaal, Norway*
[3] *Department of Anesthesiology and Intensive Care, Haukeland Univeristy Hospital, Norway*
[4] *Faculty of medicine, University of Oslo, Norway*
[5] *Department of Molecular Biosciences, University of Oslo, Norway*

Background/aims: The μ-opioid receptor (OPRM1) is the major target of endogenous opioid peptides and opioid analgesic agents. An important single nucleotide polymorphism (SNP) in this gene is the functional SNP, rs1799971, leading to a substitution of asparagine (Asn) to aspartic acid (Asp) at codon 40 in exon 1. Previous studies have suggested that this SNP may give different phenotypes in males and females. In the present study we therefore investigated whether the OPRM1 Asn40Asp SNP, grouped by gender, could predict clinical outcome regarding progression of pain intensity and disability in patients with discogenic low back pain and sciatica.

Methods: Patients ($n = 252$) with lumbar disc herniation and sciatic pain, all European-caucasian, were recruited from Oslo University Hospital Ullevål and Haukeland University Hospital. Blood samples were drawn, genomic DNA isolated and the OPRM1 Asn40Asp SNP was detected by TaqMan methodology. Pain intensity and functional consequences were rated on a visual analogue scale (VAS), by McGill Sensory and by Oswestry Disability Index (ODI) over a 12 months period (inclusion, 6 weeks, 6 months and 12 months).

Results: The genotype */Asp was associated with more pain in women, but seemed to protect the men from pain after lumbar disc herniation. Wildtype Asn/Asn women and men reported similar pain ratings. Analysis of the recovery for the four groups; women Asn/Asn, women */Asp, men Asn/Asn and men */Asp, showed that the */Asp women had a significantly slower recovery, i.e., pain intensity over time than the */Asp men (VAS activity score $p = 0.002$, McGill sense score $p = 0.021$, ODI $p = 0.205$, rmANOVA including covariates smoke, treatment and age with $p \leq 0.1$).

Conclusion: The present data suggest that the OPRM1 Asp variant increases the pain intensity in women, but have the opposite effect on men the first year after a disc herniation.

http://dx.doi.org/10.1016/j.sjpain.2012.05.025