GENE EXPRESSION PROFILING OF CUTANEOUS INJURED AND NON-INJURED NOCICEPTORS IN SNI ANIMAL MODEL OF NEUROPATHIC PAIN

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Supplementary Figures 1-3









Supplementary Fig. 1: Definition of the cell size criteria for the LCM of nociceptors. Immunohistochemistry of fresh DRG tissue for (A) nociceptor marker peripherin, (B) large-sized neuron marker NF-200 and (C) merged figures (scale bars = 100μ m). Analysis of average cross-sectional area for (D) peripherin and (E) NF-200 in L4-L5 sections issued from 1 week SNI or sham animals. To note no significant differences were detected between the two surgeries. (F) Analysis of the cross-sectional area distribution for the peripherin- and NF-200-positive neurons reveal an optimal cut off cross-sectional area of 500 μ m², within 90% of neurons were peripherin positive and only 5% were NF-200 positive.







Supplementary Fig. 2: Expression of somatosensation markers on microarray analysis from LCM samples. (A) Analysis of transcript levels for known somatosensory mediators (list modified from Chiu et al. (ref)) in LCM samples from sham condition shows the presence of several markers for thermosensation and nociception. (B) Scatter plots of top 5% genes on the microarrays for the sham condition reveal the high expression of typical nociceptive markers, such as Calca (CGRP), Scn11a (Nav1.9) and Trpv1.

<u>Up-regula</u>	ated genes	
Fold	Non-injured	Injured
> 2	15	422
> 5	3	91
> 10	0	33
<u>Down-reg</u>	gulated genes	
Fold	Non-injured	Injured
> 2	6	180
> 5	0	7
> 10	0	0

Supplementary Fig. 3: Fold change of regulated gene. Number of up-regulated and down-regulated genes per

conditions compared to sham and classified by fold change (for statistic see method section).