**Prdm12 deficiency impairs sensory nervous system function and organization**

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**Introduction**

PRDM12 is a member of a larger family of PR-domain containing transcriptional regulators. PRDM12 engages with DNA and G9a methyl-transferase to ultimately dictate cell-fate decisions during early sensory neuronal development. Mutation in any part of PRDM12 gene causes complete insensitivity to acute and chronic pain in humans, a condition named Congenital Insensitivity to Pain (CIP). These patients also suffer from recurrent skin infections, most commonly caused by *S. aureus*. In order to investigate molecular background of the observed phenotype, several conditional knockout mouse models have been developed and phenotyped on a behavioral, microanatomical and electrophysiological level.

**Objective:** Exploring behavioral, (micro)anatomical and electrophysiological phenotype of Prdm12-deficient murine models of Congenital Insensitivity to Pain.

**Goals:**
1. Behavioral phenotyping of the developed conditional knockout murine models
2. Microanatomical phenotyping of the sensory nervous system in Prdm12-deficiency
3. Electrophysiological properties of individual Prdm12-deficient nociceptors

**Electrophysiological phenotype**

![Single cell electrophysiological studies](image)

**Conclusions**

1. **Avil-Cre;Prdm12fl/fl** and **R26CreER^{II};Prdm12fl/fl** conditional knockout models phenocopy behavioral phenotype of insensitivity to pain.
2. Nociceptors in developmental advilin **Prdm12-deficient** model show impaired innervation of the skin and absence of sensory fibers in the epidermis.
3. **Prdm12-deficient** nociceptors show impaired electrophysiological properties, suggesting impaired voltage-gated sodium channels.
