

Selective Inhibition of ERK2 for Persistent Inflammatory and Neuropathic Pain

Lead Inventor:

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Background & Unmet Need

- Neuropathic pain is caused by neural damage or disease that affects the somatosensory system (e.g., diabetic peripheral neuropathy)
- Inflammatory pain is caused by injury or diseaseinduced release of inflammatory mediators (e.g., rheumatoid arthritis)
- The extracellular signal-regulated kinase 1 (ERK1) and ERK2 are evolutionary conserved serine/threonine protein kinases
- ERK1/2 signaling in spinal cord dorsal horn (SCDH) has been implicated in injury-induced pain hypersensitivity
- Unmet Need: Novel therapeutic approaches for long-term, safe control of pain

Technology Overview

- The Technology: Methods and compositions for treating pain by administering a neurotropic recombinant adeno-associated virus (rAAV) encoding a nucleic acid inhibitor of ERK2 expression
- Discovery: Selective inhibition of ERK2 expression in neurons of the SCDH in mice with a small interfering RNA (siRNA) protects against pain
- PoC Data: The ERK2 siRNA protected mice against mechanical allodynia and thermal hypersensitivity following the induction of peripheral inflammation in the hind paw
- Findings demonstrate that ERK2 in SCDH neurons is critical for signaling the development of pain hypersensitivity and that ERK2 inhibition can reduce or inhibit pain transmission in the CNS

Inventors:

Charles E. Inturrisi Sandra M. Garraway Qinghao Xu

Patents:

US Patent 8,951,979

Publications:

Xu et al. J Neurosci. 2008.

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Cornell Reference:

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Technology Applications

- Treatment and prevention of neuropathic and inflammatory pain
- Research tool for siRNA inhibition of ERK2 to foster additional studies on the mechanisms of pain

Technology Advantages

- Unlike opioids, this approach is not associated with the development of tolerance, overdose, or abuse
- Delivery of the siRNA by an adeno-associated viral vector has the potential to deliver long-term pain relief

Supporting Data / Figures

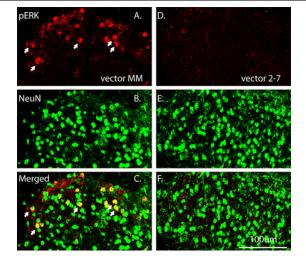


Figure 1: pERK1/2 immunolabeling was significantly reduced by vector 2-7 compared with the control vector MM at 3 weeks after rAAV vector administration.

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