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Antisense oligonucleotide therapeutic approach for chronic pain disorders

Stanford researchers have developed a targeted antisense oligonucleotide (ASO) therapy to selectively reduce the expression of NaV1.7, a key sodium channel implicated in chronic pain signaling.

Chronic pain affects millions of people worldwide and is often treated with opioids or other systemic drugs that carry significant risks, including addiction and limited long-term effectiveness. As the need for safer, more targeted therapies grows, researchers are turning to non-opioid strategies that address the underlying biology of pain. One of the most promising targets is NaV1.7, a sodium channel that plays a central role in transmitting pain signals. However, efforts to block NaV1.7 directly have faced major challenges due to delivery issues and limited target specificity.

To overcome these limitations, Stanford researchers developed a gene-targeted approach using ASOs to reduce NaV1.7 expression at the mRNA level with high specificity. Using human somatosensory organoids, the team demonstrated a significant and specific reduction in SCN9A mRNA and NaV1.7 protein levels, which resulted in decreased spontaneous neuronal activity without affecting other sodium channels. The result is a less excitable pain-transmitting neural circuit, offering a promising non-opioid treatment for chronic pain.

This platform establishes a new direction for precision pain therapeutics by combining disease-relevant human models with gene-targeted ASO strategies. It provides highly specific and scalable interventions for a range of chronic pain disorders, including neuropathic and inflammatory pain conditions.

Applications

- Targeted therapy for chronic pain disorders, including neuropathic and inflammatory pain
- Precision modulation of NaV1.7 for non-opioid pain management strategies
- Preclinical screening of ASO-based pain therapeutics

Advantages

- Selectively downregulates SCN9A to reduce NaV1.7 expression in human sensory neurons
- Reduces neuronal excitability without off-target effects on other sodium channels
- Non-opioid mechanism with potential for peripheral delivery
- Scalable platform for diverse chronic pain indications

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