An Analysis of the NaV1 VGSC Family to Develop a Treatment for Nociceptive Pain

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The opioid crisis is a deeply rooted issue in our society today that must be addressed. 75% of people who began abusing opioids in the 2000s started with prescription drugs. To address this, I worked to reject the null hypothesis that there exists no relationship between gene expression and nociception and enable me to develop an epigenetic pain treatment. I hope to help fight the opioid crisis by reducing the number of patients who become addicted to prescription opioids. Existing data show that the NaV1 VGSC family plays a crucial role in affecting nociception and loss of function in NaV1 can lead to pain insensitivity. I analyzed NCBI dataset GSE61373 through GEO2R, STRING-dB, GeneCards, Linux, and R to analyze samples from wild type mice and mice with NaV1 channels knocked out. Genes such as KCND1 and BANK1 were overexpressed in wild type mice, making them a target for regulation. I was able to reject my null hypothesis by establishing a relationship between gene expression and sensitivity to pain. This study showed that epigenetic pain therapy can be used to decrease sensitivity to pain. As an epigenetic therapy, transcription factors can decrease expression of the KCND1 and BANK1 genes to reduce nociception in the human body. Compared to current pain treatment options, primarily painkillers, epigenetic therapy is much less invasive, and reduces pain at its root. This solution offers much promise in the realm of novel pain treatments. Further research would be clinical trials of this therapy.

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