PUBLIC ABSTRACT

Pain is a common sensation that everyone experiences and is a critical warning system used to protect our body from damage. Our understanding of pain has advanced to a great degree in the past decades, but, unfortunately this increase in understanding has not resulted in better therapies for all types of pain.

Pain associated with cancer is among the most painful pathological conditions someone could experience. How and why this pain is so excruciating is not well understood. However, the prevailing hypothesis is that the cancer cells can release factors that can sensitize adjacent pain-sensing nerve fibers. My results show an important factor for cancer cell growth, parathyroid hormone-related peptide (PTHrP), can sensitize the critical pain-sensing receptor, transient receptor potential vanilloid 1 (TRPV1) on sensory neurons. TRPV1 is important for inflammatory pain and has previously been implicated as an important component of cancer associated pain in some animal models. Additionally, I show that PTHrP-mediated sensitization of TRPV1 causes increase in pain behaviors in mice.

Overall this research indicates that both PTHrP and TRPV1 are potential pharmacological targets for in the treatment of cancer pain. However, further research is needed to confirm these targets before it could reach the clinic. In a broader sense, this research further implicates TRPV1 as a possible mediator of cancer pain.