

Local Effect of IB4+ Neurons in Carcinogenesis and Pain

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Cancer patients commonly experience pain which continues to be one of the most challenging symptoms to treat and manage. Subsets of nociceptors that are responsible for cancer-induced pain remain to be fully understood. One way to classify nociceptors is based on their ability to bind the plant glycoprotein lectin IB4. Recent study suggested that μ and δ opioid receptors are differentially expressed on IB4 (-) and IB4 (+) nociceptors, which control thermal and mechanical pain, respectively. This study aimed to examine the role which IB4(+) neurons play in regulating cancer induced mechanical pain. Moreover, the effect of these sensory neurons in carcinoma growth was also investigated. Selective removal of IB4(+) nociceptors using IB4-saporin (IB4-SAP) conjugates showed significantly decreased cancer induced mechanical allodynia during late-stage cancer (25 days post cancer supernatant injection), as measured by increased paw withdrawal thresholds to mechanical stimulation. However, IB4-SAP treatment also had a significant effect on cancer induced thermal hypersensitivity, as shown by decreased paw withdrawal thresholds to thermal stimulation. Moreover, the treatment did not show any significant effect in carcinoma growth, as the tumor size showed similar trends in both the IB4-SAP and the control groups. We will further investigate the effect of thermal hypersensitivity by antagonizing TrpV1 receptor, which is expressed in IB4(-) neurons.