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Modulation of neuroimmune interactions using nanotechnology to prevent the development of chronic pain

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The interactions of glial cells (spinal cord) or macrophages (periphery) and neurons have been demonstrated to be significant players in the mechanisms underlying chronic pain. We have identified novel signaling pathways that physiologically regulate mitogen activated-protein kinases (MAPKs), namely MAP kinase phosphatases (MKPs) and CD163. We uncovered that both molecules seem to play significant roles in microglial cells in the spinal cord, and in macrophages at the periphery in regulating the resolution of inflammatory processes. We have observed that the lack of MKP-3 (KO mice) in spinal cord or the periphery results in the development of chronic pain following a surgical model of acute pain. Likewise, we have observed that the induction of MKPs or CD163 using nanotechnology in microglia or macrophages prevent the development of chronic pain, and induces an anti-inflammatory phenotype by reducing the expression of p-p38 and p-ERK MAPKs. Our data uncovered that MKPs or CD163 molecules are suitable targets for gene therapies directed to prevent the development of chronic pain. Furthermore, we have demonstrated that specific nanoparticles with proven clinical relevance could be implemented to develop an innovative cell-directed gene therapy for chronic pain conditions.

Biography

E Alfonso Romero-Sandoval is originally from Guatemala, and received his MD from Universidad de San Carlos de Guatemala in 1999 and his PhD in Neuroscience from Universidad de Alcalá, Alcalá de Henares, Spain, in 2003. He did a postdoctoral training at Wake Forest University, Winston Salem, NC (2003-2006) and at Geisel Dartmouth Medical School, Lebanon, NH (2006-2007). He was Instructor (2007-2009) and Assistant Professor (2009-2012) at Geisel Dartmouth Medical School, Lebanon, NH. Currently (2012-present) he is Associate Professor and Director of Research at Presbyterian College School of Pharmacy, Clinton, SC. Romero-Sandoval is studying the molecular mechanisms of spinal cannabinoid receptor 2 activation for induction of analgesia, the role of endocannabinoids in postoperative pain, the function of phosphatases and kinases in spinal cord in the transition from acute to chronic pain, the use of nanotechnology to promote surgical wound healing and to prevent the development of chronic postoperative pain.

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