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Novel mechanism-based targets for pain treatment

Kalpna Gupta
University of Minnesota, USA

Mast cells are tissue resident granulocytes known for their role in itch and anaphylaxis. We examined the contribution of mast cell activation in chronic and acute pain. We used homozygous BERK sickle mice that have constitutive chronic pain and hypoxia/re-oxygenation evoked acute pain. These mice mimic the features of clinical sickle cell disease (SCD), which is accompanied by severe chronic pain and recurrent episodes of acute pain. We found that mast cell degranulation/activation is significantly higher in the skin of sickle mice as compared to controls. This increased mast cell activation contributes to promoting neurogenic inflammation and nociceptor activation via the release of tryptase and substance P in the skin and dorsal root ganglion. Inhibition of mast cells with imatinib *in vivo*, led to a significant decrease in the release of cytokines from skin biopsies *ex-vivo*. Importantly, it led to a correlative decrease between GMCSF and white blood cell counts in sickle mice. Mast cell deletion in sickle mice as well as pharmacologic treatment with imatinib led to a decrease in tonic and hypoxia/re-oxygenation evoked acute hyperalgesia in sickle mice. Mast cell stabilizer cromolyn sodium reduced chronic hyperalgesia and improved the outcome of relatively lower dose of morphine, which is otherwise ineffective. We conclude that mast cells provide a druggable target to ameliorate sickle pathophysiology and pain.

gupta014@umn.edu

G-RMPP: Gait retraining as management for patellofemoral pain syndrome

Jenevieve Roper
California State University, USA

Gait retraining is a newly researched method for management of patellofemoral pain (PFP). Patellofemoral pain, more commonly known as anterior knee pain, is a common running ailment that typically affects more women than men. Although it affects many individuals, the cause is relatively unclear. Researchers agree that the cause is likely multifactorial with several perturbations leading to the development of PFP. Of those, it appears that patellofemoral joint stress (PFS) has a strong association with PFP. Therefore, a reduction in PFS is thought to lead to reduced PFP. Several interventions have been investigated with their ability to reduce PFP. Most of this research focused on hip kinetics and kinematics, showing some success in reducing PFP. However, a recent study indicated that perturbations at the hip may be a compensatory mechanism that individuals develop to manage the pain and symptoms. New research on foot strike patterns have shown that rear-foot strike running is associated with greater PFS compared to forefoot strike running. Subsequently, it was demonstrated that a significant reduction in PFP occurs as a result of switching foot strike patterns in runners affected by PFP. Specifically, changing from rear-foot strike running to fore-foot strike running has led to reductions in PFS, patellofemoral contact force, knee abduction and PFP, suggesting that it is an effective intervention for management of PFP. It is worth noting that the change in foot strike pattern increased Achilles tendon force, which potentially increases the risk of injury at the ankle.

Jenevieve.Roper@csusb.edu